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ECOLOGICAL RISK ASSESSMENT  
MONTROSE CHEMICAL CORPORATION  
LOS ANGELES, CALIFORNIA

Draft Report  
CH2M HILL  
Oakland, California

# TES 12

Technical Enforcement Support  
at Hazardous Waste Sites  
Zone IV  
Regions 8, 9, and 10

**PRC**

PRC Environmental Management, Inc.



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
REGION IX  
75 Hawthorne Street  
San Francisco, CA 94105

NOV 03 1992

MEMORANDUM

SUBJECT: Transmittal for review of CH2MHill preliminary ecological risk assessment of the Montrose Superfund site

FROM: Bruce A. Macler, Regional Toxicologist  
Water Management Division, W-6-1 *Baum*

TO: Montrose ecological assessment workgroup  
(See attached list)

Enclosed is a copy of the preliminary draft ecological risk assessment for the Montrose Chemical Superfund site for your review. I would appreciate your written comments back to me by December 1st. We would like to hold a meeting to discuss this ecological risk assessment and comments on Tuesday, December 15th. Following this meeting, we will formally respond to comments and reach a decision.

Note that the majority of data from the studies prior to CH2MHill's assessment was invalidated. New studies will provide sediment and surface water data, but will not be available until about October, 1993.

I am particularly interested in your answers to the following questions:

- 1) Does this assessment adequately describe the ecological situation at the Montrose site? If not, will it be sufficient when validated sediment and surface water contamination data are available? Do we need to do further work to provide an adequate description?
- 2) Does the assessment contain sufficient information to determine whether a detrimental ecological effect resulted from DDT and other contaminants from the Montrose Superfund site? If no, is the evidence adequate to support your view, or is more evidence necessary?
- 3) If you believe additional studies are necessary, are those proposed by CH2MHill the appropriate ones, or are others necessary? Please specifically describe any additional studies you believe to be necessary.

Thank you very much for your attention to this. If you have questions, please call me at 415 744-1884.



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
REGION IX  
75 Hawthorne Street  
San Francisco, CA 94105

MEMORANDUM

November 11, 1992

Subject: Distribution list for reviewers of the Montrose Ecological Risk Assessment

From: Bruce A. Macler, Regional Toxicologist  
Water Management Division, W-6-1

To: Montrose Ecological Risk Assessment Workgroup

The distribution list for the Draft Montrose Ecological Risk Assessment was inadvertently omitted when the report was distributed for review. Attached is that distribution list for the Montrose Ecological Risk Assessment Workgroup. Please call Nancy Woo (415-744-2394) or Bruce Macler (415-744-1855) of U.S. EPA Region IX if you have questions. We look forward to receiving your review comments on the Ecological Risk Assessment by December 1, 1992.

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MONTROSE CHEMICAL CORPORATION  
LOS ANGELES, CALIFORNIA**

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CH2M HILL  
Oakland, California**

**Prepared for**

**U.S. ENVIRONMENTAL PROTECTION AGENCY  
Office of Waste Programs Enforcement  
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## Acronyms and Abbreviations

1,1,1-TCA	1,1,1-trichloroethane
1,2-DCA	1,2-dichloroethane
1,2-DCE	1,2-dichloroethene
ASTER	Assessment Tools for the Evaluation of Risk Ecotoxicity Profiles
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	bioconcentration factor
BEIA	Biomedical and Environmental Information Analysis
bgs	below ground surface
BHC	benzene hexachloride
CDFG	California Department of Fish and Game
CEC	California Energy Commission
CNDDB	California Natural Diversity Data Base
DDD	1,1-dichloro-2,2-bis( <i>p</i> -chlorophenyl)ethane
DDE	1,1-dichloro-2,2-bis( <i>p</i> -chlorophenyl)ethene
DDT	1,1,1-trichloro-2,2-bis( <i>p</i> -chlorophenyl)ethane
DNAPLs	dense nonaqueous phase liquids
DOJ	Department of Justice
E&E	Ecology & Environment
EC	Effective Concentration
EPA	U.S. Environmental Protection Agency
ER-M	Effects range-median
ER-L	Effects range-low
H+A	Hargis + Associates
HSDB	Hazardous Substances Data Bank (Data Base)
JWPCP	Joint Water Pollution Control Plant
LACDPW	Los Angeles County Department of Public Works
LADWP	Los Angeles Department of Water and Power
LACFCD	Los Angeles County Flood Control District
LC	Lethal Concentration
LD	Lethal Dose
LNAPLs	light nonaqueous phase liquids
LOAEL	Lowest observed adverse effect levels
LOELs	Lowest observed effect levels
M&E	Metcalf and Eddy
NIOSH	National Institute for Occupational Safety and Health
NOAA	National Oceanic and Atmospheric Administration
NOAEL	No observed adverse effect levels
NPL	National Priorities List
NRC	National Research Council
NS&T	National Status and Trends
p-CBSA	parachlorobenzene sulfonic acid
PCB	polychlorinated biphenyl

PCE	tetrachloroethene, or perchloroethene
PHRED	Public Health Risk Evaluation Database
POLA	Port of Los Angeles
ppt	parts per thousand
RI	remedial investigation
RTECS	Registry of Toxic Effects of Chemical Substances (Data Base)
RWQCB	Regional Water Quality Control Board
SCAQMD	South Coast Air Quality Management District
SCCWRPA	Southern California Coastal Water Research Project Authority
SPRR	Southern Pacific Railroad
TCE	trichloroethylene
TDB	Toxicology Data Bank
TIC	tentatively identified compound
TOXNET	TOXicity Data NETwork (Data Base)
VOC	volatile organic compound
WHO	World Health Organization
WHR	Wildlife Habitats Relationships (Data Base)

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## 1 Introduction

## Section 1

### Introduction

From 1947 to 1982, Montrose Chemical Corporation of California (Montrose) owned and operated a dichlorodiphenyltrichloroethane (DDT) manufacturing facility in Los Angeles, California. DDT was used throughout the world as a mosquito vector control for the eradication of malaria (Meister, 1990). However, because of environmental and human health concerns associated with DDT, all uses in the U.S., except emergency public health uses, were canceled effective January 1, 1973 (EPA, 1975; Meister, 1990). Production of DDT for export was allowed until 1982, when it was also banned. Operation of the Montrose plant was discontinued in 1982 and the facility was dismantled in 1983 (Ecology and Environment, Inc. [E&E], 1986). In April 1985, Montrose regraded and capped most of the property with asphalt. The property is currently unoccupied and fenced.

DDT is a chlorinated insecticide known to be persistent (does not degrade readily) in soil. Degradation of DDT is very slow, with "half-life" values of 10 years or more. In aquatic ecosystems, DDT and its metabolites are found in water, aquatic life, and particularly in sediments. DDT and its metabolites, DDE and DDD, have entered the food chain of mammals, birds, fishes, and other animals. DDT is concentrated by freshwater and marine plankton, insects, molluscs, and other invertebrates and fish, and is transferred through the aquatic foodchain. Effects on phytoplankton species composition include decrease in photosynthesis and growth rates, resulting in upset to the balance of the aquatic ecosystem. In molluscs and arthropods, DDT can result in lethal effects including starvation in invertebrate predators, and sublethal effects such as reproductive failure. In fish, DDT has been shown to reduce reproductive success and upset various biochemical systems (EPA, 1975).

DDT also affects the terrestrial ecosystem where it is concentrated in and transferred through invertebrates, mammals, birds, reptiles, and amphibians. Effects have included decreases of eggshell thickness in birds, particularly those feeding in fresh and brackish waters, resulting in impaired reproductive success (EPA, 1975).

## 1.1 Overview of Report

This document presents a preliminary ecological risk assessment of areas potentially affected by releases from the Montrose Chemical Corporation of California property, located in Los Angeles, California. Available literature was used to identify chemicals of concern associated with Montrose operations, provide analytical data for initial assessment of environmental impact, and characterize habitats potentially affected by releases from the Montrose property.

This ecological risk assessment was conducted in accordance with EPA *Risk Assessment Guidance for Superfund—Volume II, Environmental Evaluation Manual* (EPA, 1989). The scope of the assessment was developed in accordance with supplementary guidance including *Developing a Work Scope for Ecological Assessments*, ECO Update, Volume 1, No. 4 (EPA, 1992), and is presented in *Work Scope for Ecological Risk Assessment, Montrose Chemical Corporation, Torrance, California* (CH2M HILL, 1992).

In accordance with guidance (EPA, 1992), a phased approach was adopted for this investigation to ensure that the level of effort expended met the needs of characterizing impacts, but did not exceed those needs. With the phased approach, data or observations from one phase determine whether further studies are needed to meet the assessment's objectives, and, if so, to define the subsequent studies. Interim reports, such as this, are provided for review by the U.S. EPA Remedial Project Manager (RPM), and additional tasks are performed if authorized by the RPM. This ecological risk assessment presents the findings of the first phase of a complete ecological risk assessment.

## 1.2 Scope of Investigation

A Phase I ecological risk assessment was conducted to address the Montrose property and surrounding areas. The scope of the investigation was limited to those areas potentially affected by environmental releases of chemicals of concern through surface drainage and atmospheric transport. These areas are defined in Section 2. Impacts associated with releases of chemicals of concern through the sanitary sewer system or offshore dumping were not addressed; assessments associated with these activities are being conducted separately by state and federal Natural Resource Trustees.

Existing literature and reports were reviewed to provide information on the property history, data addressing chemicals of concern, characterization of habitat, and documentation of effects associated with releases from Montrose. In addition to a review of available literature, two reconnaissance-level surveys of the area were conducted to provide a preliminary evaluation of habitat and ecological receptors to determine the adequacy of literature characterization, evaluate habitat suitability, qualitatively identify evidence of stress, and note the presence or absence of special-status species or other species of concern.

In addition, a preliminary exposure assessment and risk characterization are presented. These are preliminary in that they use only data available in literature; no samples were collected for this effort. A discussion of the fate and transport potential of the chemicals of concern, as it affects the potential for exposure, is also included. This preliminary exposure assessment is intended to identify the potential for adverse effects to habitats potentially affected by releases from Montrose. Based on the preliminary risk assessment, recommendations are made as to the need to conduct further studies and to identify additional data needed to conduct them.

### **1.3 General Assumptions**

This preliminary assessment uses previously reported analytical data for sediment, soil, and water concentrations. Data used are assumed to be valid and useable except where presented in this document with qualifiers. In addition, all results presented are assumed to be quantitatively accurate, even though analytical methods have changed and improved over time. The risk characterization is based primarily on the most recent available data.

### **1.4 Organization**

A summary of existing data and information gathered during two reconnaissance surveys is presented in this document. Organization of the document is as follows:

- Description of the property and surroundings, determination of areas potentially impacted by environmental releases from the property, and definition of the study area (Section 2).
- Review of the nature and extent of contamination by medium and contaminant type as presented in available literature (Section 3). This section also identifies chemicals of concern for this ecological assessment.
- Identification of potentially exposed habitats and ecological receptors (Section 4).
- Review of potential exposure mechanisms and pathways (Section 5).
- Initial toxicity assessment (Section 6).

- Preliminary risk characterization (Section 7).
- Conclusions and limitations associated with this study (Section 8).
- Recommendations for further studies (Section 9).

This document will be used as the basis for evaluating the need for, and preparing a work plan for, further studies needed to characterize actual or potential adverse effects associated with Montrose contaminants.

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## **2 Description of Property and Surrounding Area**

## Section 2

# Description of Property and Surrounding Areas

## 2.1 Setting

The Montrose Chemical Corporation of California (Montrose) owns approximately 13 acres in the City and County of Los Angeles (Figure 2-1). Figure 2-2 presents an aerial photo of the area, including the major drainage channels and highways. The Montrose property, shown on Figure 2-3, will be referred to as "the property" or as Montrose for the remainder of this document. Montrose's business address is 20201 South Normandie Avenue, Torrance, California. Approximately 1 mile northeast of the property is the intersection of the San Diego Freeway (i.e., Interstate 405) and Harbor Freeway (i.e., Interstate 110). The City of Torrance is located west of the property beyond Western Avenue. Other surrounding cities include the City of Carson located east of the Harbor Freeway, and Gardena located about 1.5 miles north of the property beyond the San Diego Freeway. A portion of Los Angeles County is east of the property.

The area immediately surrounding the Montrose property is zoned industrial; commercial and residential areas exist within a 1-mile radius of the property. Bordering the property to the east are Normandie Avenue (Figure 2-3) and the Southern Pacific Railroad right-of-way. Southwest of the property is the Jones Chemical Company. The Los Angeles Department of Water and Power's (LADWP) 100-foot-wide easement runs east-west, to the south of both the Montrose and Jones Chemical properties. Beyond the LADWP right-of-way, south of 203rd Street, is the Farmer Brothers Coffee Company. The McDonnell Douglas Corporation property occupies the remainder of the block to the north and west of the Montrose property. Across Normandie Avenue to the east is the Del Amo Superfund Site, the former location of a butadiene-styrene rubber manufacturer. Portions of the Del Amo site are currently used for

manufacturing facilities such as the Coca Cola bottling plant. Bordering the LADWP right-of-way, south of the Del Amo site, are the nearest occupied residences.

The Montrose Superfund site ("Site") includes on-property and off-property contaminated soil, contaminated groundwater, the Normandie Avenue Ditch, the Kenwood Drain, Torrance Lateral, Dominquez Channel, and Consolidated Slip.

### **2.1.1 Property Ownership**

The Montrose DDT manufacturing and formulation facilities were situated on a 13-acre parcel of land located in Los Angeles, California. The owner of the property during the time Montrose operated its DDT facilities was Stauffer Chemical Corporation ("Stauffer"). Stauffer owned the 13-acre parcel from approximately 1943 to 1987. In 1987, Atkemix Thirty-Seven, Incorporated, acquired the 13 acre property.

There were multiple chemical-related operations on this 13-acre parcel of land and the adjacent property now occupied by the Jones Chemical Company. Montrose's DDT manufacturing and formulating operations ran from 1947 to 1982. In 1953, Stauffer constructed a Lindane pilot plant on the Montrose property. Operation of that plant ceased sometime in the 1960s. Stauffer operated a sulfuric acid plant at the adjacent property from approximately 1943 to 1952. In the same location as Stauffer's acid plant, the predecessor of Jones Chemical Corporation began its operations sometime in 1956. Jones Chemical Corporation operations have continued to the present date.

### **2.1.2 Montrose Operational History**

Montrose, formerly a subsidiary of Stauffer, has owned this facility since it was established in 1947. Montrose continues to own the property, although the property has not been active since 1982.

During its years of operation, 1947 to 1982, the Montrose property consisted of a central process area and surrounding general areas (Figure 2-4). The central process area included the DDT processing building, the adjacent processing equipment and transfer station areas, the filtration area, the waste recycling pond, and the acid recovery area and several below-grade tanks (not shown). This area was approximately 300 feet by 400 feet.

South of the central processing area was the formulation and grinding plant (installed in 1960) and warehouse (installed in 1964) in which DDT awaiting off-property transport was stored. The main offices, laboratory, warehouses, special products plant, and locker rooms were located to the east. A cooling tower, the machine shop, a truck repair facility, a storage area and lunch room, and a maintenance shop were located to the west.

Aerial photographs available from 1938 through 1983 show the locations of railroad tracks, storage facilities, and waste pits throughout the property (M&E, 1986). Storage facilities were located along the north, south, and west borders of the property. Two possible waste pits were located west of the oil storage facility. No information could be obtained on the types of waste stored in either the facilities or the pits (M&E, 1986). In addition, four railroad spurs ran along the south, east, and west perimeters of the property. The tracks running along the west and south perimeters were abandoned between 1965 and 1974.

Manufacturing of DDT occurred primarily in the central processing area. Chlorobenzene and chloral were combined in the presence of sulfuric acid to form DDT; acid waste waters also resulted from this process. The DDT and acid wastes were separated, and the acid was drawn off the bottom of the tank. Any acid remaining in the liquid DDT solution was then neutralized with a 15 percent sodium hydroxide (caustic) solution. Following neutralization, the DDT was rinsed with hot water, purified, and crystallized. The crystallized DDT was either put into 50-pound bags and stored onsite in warehouses, transferred to the grinding plant for grinding, or sent to the formulation

plant (Hargis + Associates [H + A], 1990). From the warehouses, DDT products were shipped off-property by truck or box car.

In the formulation plant and warehouse, DDT was processed into water-dispersible powder and dusting powder. The water-dispersible powder, 75 percent DDT by weight, was made by "mixing technical grade DDT with various wetting agents, dispersing agents, and amorphous silica." The mixture was blended and milled to a particle size of less than 2.7 microns using processing equipment such as ribbon blenders, hammer mills, and air mills, and packaged. The DDT dusting powder was made of 10 percent DDT mixed with talc (H + A, 1990).

By-products of the operations included a dilute sulfuric acid liquid that included parachlorobenzene sulfonic acid (p-CBSA) and excess chlorobenzene, a dilute caustic (alkaline) waste stream from the neutralization step, and rinse water used following neutralization.

The caustic waste stream was historically drained into below-grade tanks and the waste recycling pond. In 1953, Montrose began diverting the caustic wastewater from the central process area to two redwood-lined, concrete, below-grade tanks (H+A, 1990); by 1961, Montrose was reportedly diverting its caustic wastewater to sanitary sewers flowing to the Joint Water Pollution Control Plant (JWPCP) located south of the site (DOJ, NFS011, 1682), (Young et al., 1976). Discharge to the JWPCP was stopped in April 1970, and Montrose began hauling its caustic wastes to a landfill disposal site (Young et al., 1980).

Beginning in 1947, waste acid was collected and disposed of by the California Salvage Co. at "Dumpsite No. 1," 10 miles northwest of Catalina Island. In 1961, acid wastes were recovered by separating p-CBSA and chlorobenzene, and the recovered acid was resold or sent to a Class 1 disposal facility (DOJ, NFS011). In addition to ocean dumping and landfiling, Montrose reportedly also discharged acid wastes and sludges by discharging to "a Los Angeles County wastewater treatment plant" (NOAA, 1990).

The waste recycling pond received water captured from other on-property sources such as the locker room facilities, storm runoff from the central process area, and process cooling water. The pond also served as a backup holding area into which process waste from the two tanks overflowed in an emergency. The waste pond was approximately 30 feet deep, with a 10-foot freeboard around the perimeter. The pond was lined in 1970 and remained in use until the plant closed in 1982. At that time, the sludge was removed and the "concrete" pond lining was crushed (M&E, 1986). In 1985, Montrose paved most of the property without the approval of state or federal regulatory agencies.

Before 1953, DDT was manufactured using the batch process method, using six independent 1,600-gallon batch reactors. In 1953, a large holding tank was installed to receive the reaction products from all six batch reactors. This holding tank increased DDT production by allowing for continuous operation of the processing equipment (Envirologic Data, 1991). Montrose was reported to have produced approximately 1 million to 7 million pounds per month of technical grade DDT as the manufacturing process changed throughout the 35 years of plant operations (H+A, 1990).

### **2.1.3 Current Site Description**

Most of the Montrose property is capped with asphalt; an unpaved strip exists at the southeast corner along the Southern Pacific Railroad spur. The entrance to the property is at the northeast corner, along Normandie Avenue (Figure 2-5). The only structures on the property are: three asphalt raised pads constructed by Montrose in 1985 to support planned warehouses; a trailer to support remedial investigation activities; and a trailer for Kallok Enterprises Incorporated, hired by Montrose to oversee any on-property activities. A concrete curb runs along portions of the property's perimeter, which is surrounded by a chain-link fence.

## 2.2 Regional Setting

Before the 1930s, the principal land use in the vicinity of the property was agricultural. Based on a 1938 aerial photograph, the immediate area included farms, but no railroads or business developments. By 1940, industrial, commercial, and residential areas began to develop. No additional aerial photographs of this area were available until 1956, when Montrose had been in operation for 9 years. A large community based on light commerce and industry was developing in the Torrance area (H+A, 1990).

Currently, the area surrounding the Montrose property is heavily developed with industrial, commercial, and residential land uses; it is served by a well-developed infrastructure including highways, railroad services, flood control channels, sanitary and stormwater sewers, and other services, and is interspersed with parks and other open areas. Flood control channels draining the local interior lands include the Torrance Lateral, the Dominguez Channel and the Consolidated Slip (Figure 2-6). South of the site at the mouth of the Consolidated Slip is the east basin of the Los Angeles Harbor and Terminal Island.

Residential and open areas (e.g., parks, cemeteries) exist throughout the region (Figure 2-7). A new residential area is currently being developed east of the Montrose property along the west side of the San Diego Freeway. Parks within a 1.5-mile radius of the property include Victoria Park, Victoria Golf Course, and Dominguez Golf Course along the Dominguez Channel. Open lands including the former Ascot Speedway, the Goodyear Airship Field, and a large cemetery exist to the northeast. Harbor Regional Park, approximately 4 miles south of the Montrose property, encompasses a golf course, a freshwater marsh, and Harbor Lake. Other parks (not identified) include Del Amo Park, Carson Park, Calas Park, and Dolphin Park, all located in Carson City.

To the south of Montrose are oil refineries along the southern reach of the Dominguez Channel including the Arco, Chevron, and Texaco oil refineries (Figure 2-7). Champlin Petroleum Company operates a pumping field to the east of the Consolidated Slip.

## **2.3 Topography and Drainage**

### **2.3.1 Regional**

The Montrose property is located in an area referred to as the Torrance Plain, approximately 50 feet above mean sea level. Stormwater runoff from the Torrance Plain historically followed natural drainages leading south and east to the present day Los Angeles Harbor. As early as the 1920s, the City of Los Angeles had begun developing a stormwater drainage system to serve the area. The system used both natural drainages and constructed systems. To control flooding beyond the capacity of the natural drainages, the path of the former Dominguez Creek was channelized before 1930 to drain marshy areas and provide flood control. This drainage was renamed the Dominguez Channel. Figure 2-8 shows the area's 50- and 25-foot mean sea level contours, as documented on 1958 City of Los Angeles engineering maps. Channels now referred to as the Kenwood Drain and Torrance Lateral were historically natural drainages that carried surface water runoff from the Montrose area east to the Dominguez Channel. Areas to the south of Montrose, including Harbor Lake, would not have received runoff from the Montrose area, except perhaps during severe flooding.

### **2.3.2 Local**

During the years of Montrose operations, stormwater runoff from the Montrose property flowed south and east across the property toward Normandie Avenue (Figure 2-9). Water from the west side of the property reportedly flowed south along

the railroad spurs onto the Jones Chemical property, where it entered the Jones Ditch (which follows the railroad spur east toward the southeast corner of the Montrose property) or continued south to Farmer Brothers Coffee property. Runoff from the east side of Montrose flowed to the southeast corner, under the fence, into a culvert under the railroad spur, and then into an unlined ditch referred to as the Normandie Avenue Ditch. The Normandie Avenue Ditch flowed south until diverted onto the Farmer Brothers Coffee property through a small opening in a wall; surface water then flowed onto the Farmer Brothers parking lot and to a catchbasin for a subsurface storm drain (exact location unknown).

In 1982, before most of the Montrose property was capped, surface water runoff and sediments were observed flowing south, to the catchbasin on the Farmer Brothers Coffee facility; this catchbasin reportedly entered the local stormwater system. Sampled runoff was found to contain high concentrations of DDT and chlorobenzene (E&E, 1986; Montrose, 1981). In 1983, Montrose built an earthen berm around the south and east boundary of the property to prevent runoff.

In 1985, most of the Montrose property was graded, contoured for three building pads (Figure 2-5), and covered with asphalt. The asphalt was intended to prevent soil transport via air or surface water runoff, and to minimize infiltration, while preparing the property for development of a commercial warehouse facility. The property and asphalt were graded to direct surface water runoff toward the southeast corner of the property and into the Normandie Avenue Ditch. A concrete curb was also constructed to minimize surface water running onto the property from upgradient sources (H + A, 1990).

As a result of the grading, surface water now flows toward the southeast corner, around the elevated pads. From the southeast corner, runoff can either flow under the perimeter fence and onto the railroad spurs, or into a corrugated pipe that extends beneath the railroad spur, and then continues south in the Normandie Avenue Ditch. The Normandie Ditch is now a concrete catchment located along the west edge of

Normandie Avenue. This drainage was unlined until 1986. Drainage from the Normandie Avenue Ditch enters a catchbasin located on the west curb of Normandie Avenue adjacent to the parking area for Farmer Brothers Coffee and is transported via an underground culvert to the Kenwood Drain (Figure 2-10) (M&E, 1986).

The catchments on Normandie Avenue and the Kenwood Drain are part of the City of Los Angeles stormwater system serving the Montrose property area. The Kenwood Drain discharges into the Torrance Lateral, which then discharges to the Dominguez Channel (Figure 2-6). The Dominguez Channel is a tidal flood-control channel that extends south to the Consolidated Slip portion of the Los Angeles Harbor. Each of these drainage systems is discussed further below.

The Kenwood Drain (County of Los Angeles Public Works Projects 685 and 1250) is a buried concrete storm sewer system that drains to the south beneath Kenwood Drive and then travels east beneath 209th Street until it merges with the Torrance Lateral (Figure 2-10). The Kenwood Drain was installed in 1973, replacing a natural drainage channel, which was subsequently backfilled to grade (H+A, 1990). The Kenwood Drain varies in design, including both reinforced concrete pipe and reinforced concrete box settings (LACFCD, 1973).

The Torrance Lateral (County Projects 1153 and 1232) is a fenced, open, concrete-lined drainage with a flat bottom and tapered or vertical sides. It is dry most of the year, carrying an intermittent flow consisting of urban runoff from industrial, commercial, or residential areas and runoff from rain. The Torrance Lateral receives drainage from the area bordered by the San Diego Freeway to the north, Sepulveda Boulevard to the south (Figure 2-6), and west into the City of Torrance (LACDPW, 1991). Stormwater in the Torrance Lateral flows eastward until it merges with the Dominguez Channel. For maintenance purposes, the lateral is scraped (referred to as "invert cleaning") during May of odd-numbered years. This cleaning occurs from Western Avenue (Figure 2-6) to Dominguez Channel. Removed debris and sediments are hauled to a landfill; sediment is not chemically analyzed (Nakahara, 1992).

The Dominguez Channel (Project 688) is a flood-control drainage that bisects the Torrance Plain and discharges into the Consolidated Slip just south of the Henry Ford Avenue Bridge (Figure 2-6). Where the Dominguez Channel and Consolidated Slip meet, the Channel is approximately 15 feet deep. The Channel flows thorough areas of heavy industrial development and residential areas. The Torrance Lateral enters the Dominguez Channel approximately 5.5 miles upstream of the Consolidated Slip. The Channel is tidally influenced as far north as its intersection with Vermont Avenue. The reach below Vermont Avenue consists of stone revetments or riprap and is lined on the bottom with a 5-foot-thick compacted clay lining to prevent shallow groundwater (underground freshwater) from mixing with saline water. This lining improvement was completed in 1962 (Montgomery Research, Inc., 1967). The upper reach of the Dominguez Channel northwest of Vermont Avenue is a narrower channel that has a concrete bottom and concrete revetments. The Dominguez Channel has not been dredged or scraped since 1984; information on maintenance before 1984 was not available (Nakahara, 1992). Historical records show that as early as the 1920s and 1930s, the water quality in the channel and the harbor were being affected by domestic and industrial waste discharges (Hertel, 1969). As the area became industrialized during WWII discharges to the Dominguez Channel were fouling the waterway (LACFCD, 1967). Historically, during dry weather periods there was no flow in the Dominguez Channel, except industrial wastewater and minor amounts of drainage water. The available dissolved oxygen was at low enough concentrations then ( $\leq 0.5$  mg/L) that it inhibited the growth of marine organisms that attacked harbor facilities (Hertel, 1966).

In 1951, the Dominguez Channel was described as a "filthy combination of storm drain and industrial sewer" (CDFG, 1951). In the 1960s, the California Regional Water Quality Control Board (RWQCB) and other agencies began a program to improve the water quality through controlling discharged materials and issuing discharge permits. In November of 1966, the Los Angeles RWQCB resolved to prevent nuisance conditions (odor or unsightliness), and to protect the waters of the Dominguez Channel for flood control and boating (secondary purposes). By this time, sanitary sewage was being

diverted to the local sanitary sewer systems; only industrial wastes, such as cooling tower blowdown, were released to the Dominguez Channel (LACFCD, 1967).

In February 1970, the RWQCB identified fish resources to be a beneficial use of the water in the Inner Harbor of the Port of Los Angeles; it thereby became illegal to discharge water into the Dominguez Channel that would be lethal to fish downstream. By October of 1970, County Engineer reports documented fish species in the lower regions of the Dominguez Channel, "presumably from natural migration from the harbor" (Johnson, 1970).

The Consolidated Slip begins where inflow enters from the Dominguez Channel at the Henry Ford Avenue Bridge, and ends at a constriction at the north end of the east basin where it joins the Los Angeles Harbor (Figure 2-6). The Consolidated Slip is approximately 3,000 feet long, 300 to 350 feet wide, and approximately 25 feet deep in the center. It serves as a recreational marina and a commercial port (H+A, 1990). The Slip has not been dredged since 1984; before then it was owned by Union Pacific, and the dredging history was not available (Richter, 1992).

## 2.4 Climate

The climate of the area is mediated by the Pacific Ocean. Daily weather patterns consist of night and morning low clouds followed by sunny afternoons. Another characteristic is the smog that prevails throughout the West Coast Basin (Envirologic Data, 1991). Average daily temperatures range between 55.5°F and 70.3°F (1951-1980), with annual mean temperature of 63°F (M&E, 1986). High temperatures are infrequent but occur with Santa Ana winds (Envirologic Data, 1991).

Figure 2-11 shows that prevailing winds in the area are from the west (42 percent) and west-northwest (17 percent) based on the 1981 wind rose for the Lennox area (located

approximately 6 miles northwest of the site). A leeward wind rose for the Long Beach (LGB) and Los Angeles Airports (LAX) (1965 to 1974) shows prevailing daytime winds are from the west (23 percent frequency), west-southwest (22 percent frequency at LAX), and west-northwest (10 percent frequency at LGB), with lighter winds from the east and east-northeast at LAX, and from the south at LGB (Figure 2-12). Occasionally during the fall, winter, and spring, gusty and dry northeasterly Santa Ana winds from the interior blow toward the coast. Based on the information from the wind roses, Santa Ana winds from the east occur with a 7 to 10 percent frequency.

Measurable rainfall occurs mainly from November to April (M&E, 1986; Envirologic Data, 1991). Average precipitation for the area is 12.08 inches (1951-1980). However, drought in the area for the past 5 to 6 years has decreased precipitation to below average. Data from 1988 indicated 7.96 inches of rainfall (NOAA, 1988). Occasional storm events, such as in February 1992, cause severe flooding in the area.

## 2.5 Area Hydrogeology

The Montrose property is located in the Torrance Plain of the West Coast Basin, with the El Segundo Sand Hills to the west, the Palos Verdes Hills to the southwest, and the Rosecrans and Dominguez Hills to the north and northeast, respectively. Several aquifers and aquitards exist in the quaternary- and tertiary-age marine deposits that comprise the West Coast Basin (H + A, 1990).

The four uppermost groundwater units in the area are illustrated in Figure 2-12 and include (H+A, 1990):

- **Bellflower Aquitard.** H + A have identified three distinct subunits: the Upper Bellflower Aquitard (approximately 70 to 85 feet bgs); the Bellflower Sand (approximately 110 to 140 feet bgs); and the lower

Bellflower Aquitard (approximately 140 to 160 feet bgs). Groundwater in this unit is currently not used as a source of drinking water. However, this unit may be defined by the State as a potential drinking water source.

- **Gage Aquifer.** The Gage Aquifer has a low yield and is used for groundwater supply (H + A, 1990). It is vertically confined where the Bellflower Aquitard is present, but is laterally associated with the Gardena Aquifer to the north. The Gardena Aquifer has one producing well and several other completed wells within 3 miles of the property. The Gage Aquifer extends from approximately 160 to 220 feet bgs.
- **Lynwood Aquifer.** This unit is confined to the north and east of the property. It is used for water supply by wells that are screened in both the Lynwood and the deeper Silverado Aquifers. The Lynwood Aquifer extends from approximately 250 to 300 feet bgs.
- **Silverado Aquifer.** This unit merges with the Lynwood Aquifer within 2 or 3 miles west and south of the property. Extending from approximately 350 to greater than 600 feet bgs, it is the major source of groundwater within the West Coast Basin.

Pumping and injection well activity influence the water levels and flow directions in these aquifers. A downward vertical gradient exists in each of these units because of the heavy pumping; injection wells are located and operated to prevent saltwater intrusion to the aquifers. Groundwater does not discharge to surface water because of the downward vertical gradient.

## 2.6 Natural Resources

Available reports and computerized data bases as well as two reconnaissance-level surveys were used to determine the natural resources of the area. Wildlife habitats within the area were characterized on the basis of their potential or observed use by semi-aquatic and terrestrial wildlife. Descriptions of the characteristic aquatic and terrestrial resources are provided in this section and in Section 4, Ecological Receptors.

For this project, the Wildlife Habitats Relationships (WHR) data base was used to aid in generating a list of semi-aquatic and terrestrial wildlife species potentially occurring within the area. The wildlife habitat types, as classified by Mayer and Laudenslayer (1988) for the WHR system (CDFG, 1989), include marine, estuarine, riverine, and urban habitats. The WHR is a computer data base information system created through multi-agency cooperation and is maintained by the California Department of Fish and Game (CDFG). It consists of several components used to assess vertebrate wildlife species occurrence, habitat requirements, life history information, and relative abundance.

The California Natural Diversity Data Base (CNDDDB) (CDFG, 1991) was used to identify plant and animal species of special status (i.e., state or federal threatened or endangered species or candidates for listing and other species of special concern) potentially occurring within the Montrose area and, therefore, potentially affected by discharges from the Montrose property. The CNDDDB is a computer data base that compiles records of locality, habitat, and status for sensitive species and habitats and is maintained by CDFG. Data included in the CNDDDB are compiled by opportunistic rather than systematic means and, therefore, may not include all records of species occurrences and habitats for a given area. The CNDDDB included sightings of the least tern (a federally listed endangered species) at Harbor Lake and at Belmont Shore (about 6 miles east-southeast of the Consolidated Slip).

The information obtained from both CNDDDB and WHR was used as a supplement to two reconnaissance-level surveys of the area conducted during February 26 through 28 and May 6 through 8, 1992. The primary purpose of those surveys was to determine which migratory bird species or special-status wildlife species occurred in the area. Although least terns may occur in the area (as indicated by CNDDDB), they were not observed during the reconnaissance visits. During the May survey, salinity measurements and other observations also were made to help describe the aquatic environment.

### **2.6.1 Aquatic Resources**

The aquatic resources of the area include habitats and species that are characteristic of heavily industrialized and urbanized environments in the Los Angeles region. The Dominguez Channel and Consolidated Slip provide marine and estuarine habitat that is used by numerous aquatic and semi-aquatic species, as described further in Section 4, Ecological Receptors. These species include invertebrates, fish, and birds. Although some marine mammals (i.e., seals and sea lions) could be expected to occur in this habitat, they were not observed during the surveys. Estuarine and marine habitats within Dominguez Channel extend upstream as far as Vermont Avenue, but no other wetlands are associated with upstream portions of the drainage system. Portions of the Dominguez Channel that are farther upstream from Vermont Avenue are concrete-lined; they do not provide significant habitat and are not expected to be affected by discharges from the Montrose property because of tidal gates preventing upstream flow beyond Vermont Avenue. Similarly, the Torrance Lateral is concrete-lined upstream from near the San Diego Freeway and it does not provide habitat for aquatic communities.

Freshwater aquatic habitats potentially affected by releases from the Montrose property were not identified in the area. However, some semi-aquatic species (such as treefrogs and toads) that are characteristic of such wetlands could use small or seasonal ponds

(such as in parks, golf courses, or undeveloped areas) for breeding and live in nearby terrestrial habitats within the area.

## **2.6.2 Terrestrial Resources**

The terrestrial natural resources within the area include several less intensively developed areas east of the Montrose property and north of the Torrance Lateral as well as open habitats in the Dominguez Golf Course, Victoria Golf Course, and Goodyear Airship Field, and along freeways in that vicinity (Figure 2-7).

Plants in those areas are mostly grasses and forbs, but trees and unvegetated areas also occur there and are used by wildlife. The abundance and diversity of vertebrate wildlife species in the terrestrial habitats of the area were lower than in the aquatic habitats, based on observations during the field reconnaissance surveys.

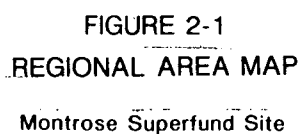
## **2.7 Areas Retained for Assessment**

On the basis of information provided in this section, areas potentially affected by releases from Montrose can be better defined. Drainages receiving surface water and sediment runoff from the property include the Jones Ditch, Normandie Avenue Ditch, Kenwood Drain, Torrance Lateral, Dominguez Channel, and Consolidated Slip. The east basin and Los Angeles Harbor, while potentially affected, are being addressed by other studies (NOAA, 1990 and 1991). Other surface water bodies, such as Harbor Lake and the associated Lomita Marsh, were not a part of the drainage system for the Montrose site area, so they were not retained as areas of concern.

Releases from the property by atmospheric transport could have affected downwind areas. Prevailing winds were documented as coming from the west, with some contribution from the west-northwest and west-southwest. Therefore, areas within

several miles to the northeast, east, and southeast were retained for assessment. Occasional Santa Ana winds were from the east. Because of prevailing wind direction, areas to the north, northwest, southwest, and south of the property (e.g., Gardena and Harbor Park areas) were not believed to have been affected, and areas near the property to the west were only infrequently exposed. However, available data documenting atmospheric releases are reviewed in Section 3. These results will be used to further define areas potentially affected by atmospheric transport of chemicals from Montrose.

In summary, areas potentially affected by releases from the Montrose property were retained for consideration in this ecological assessment, and are referred to as the areas of concern. These areas include portions of the surface water drainage system (i.e., Jones Ditch, Normandie Avenue Ditch, Kenwood Drain, Torrance Lateral, Dominguez Channel south of Vermont Avenue, and Consolidated Slip to the constriction at its southeastern end) and terrestrial habitat within 1 to 2 miles east, southeast, and northeast of the property and near the property to the west, west-southwest, and west-northwest.



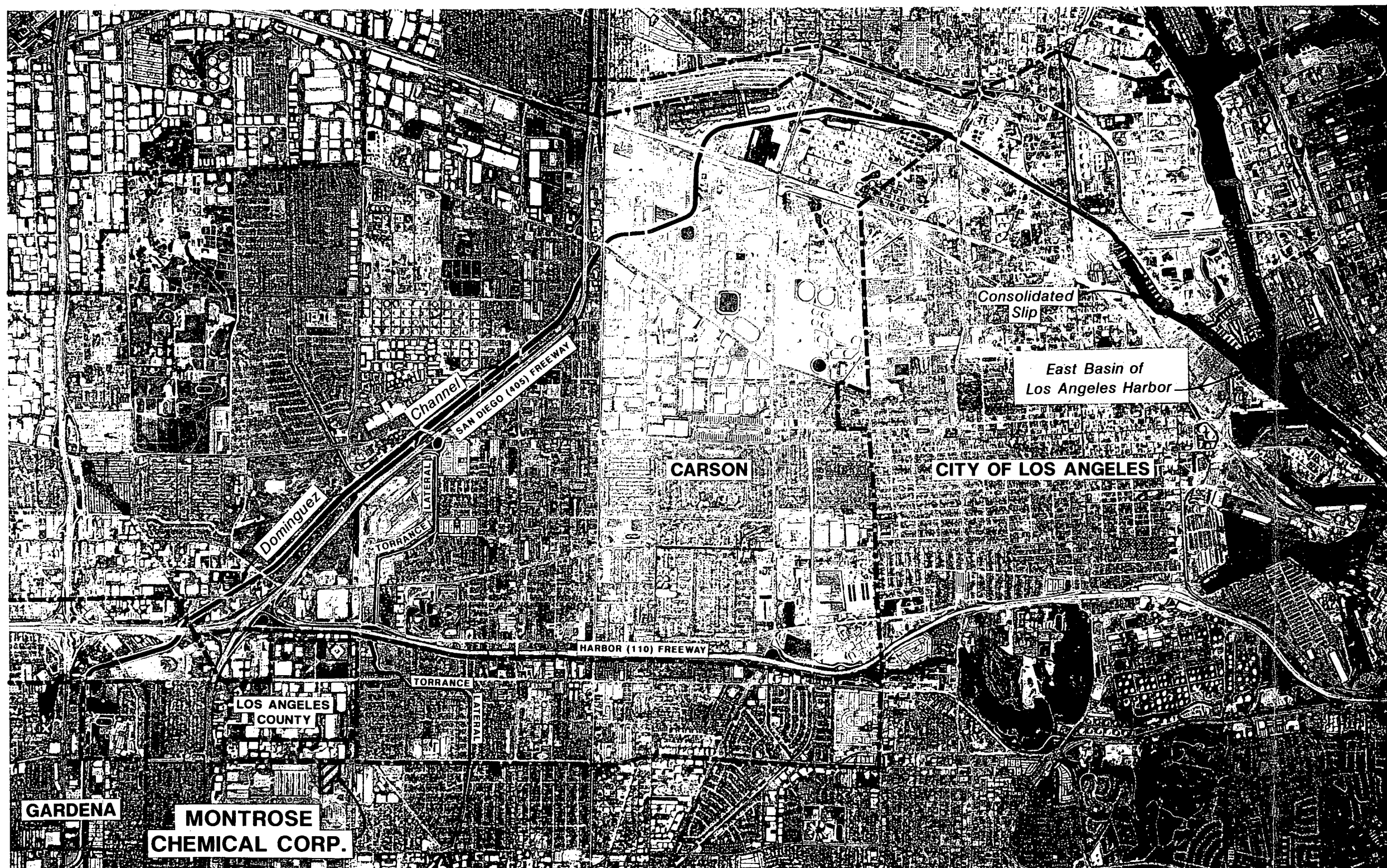
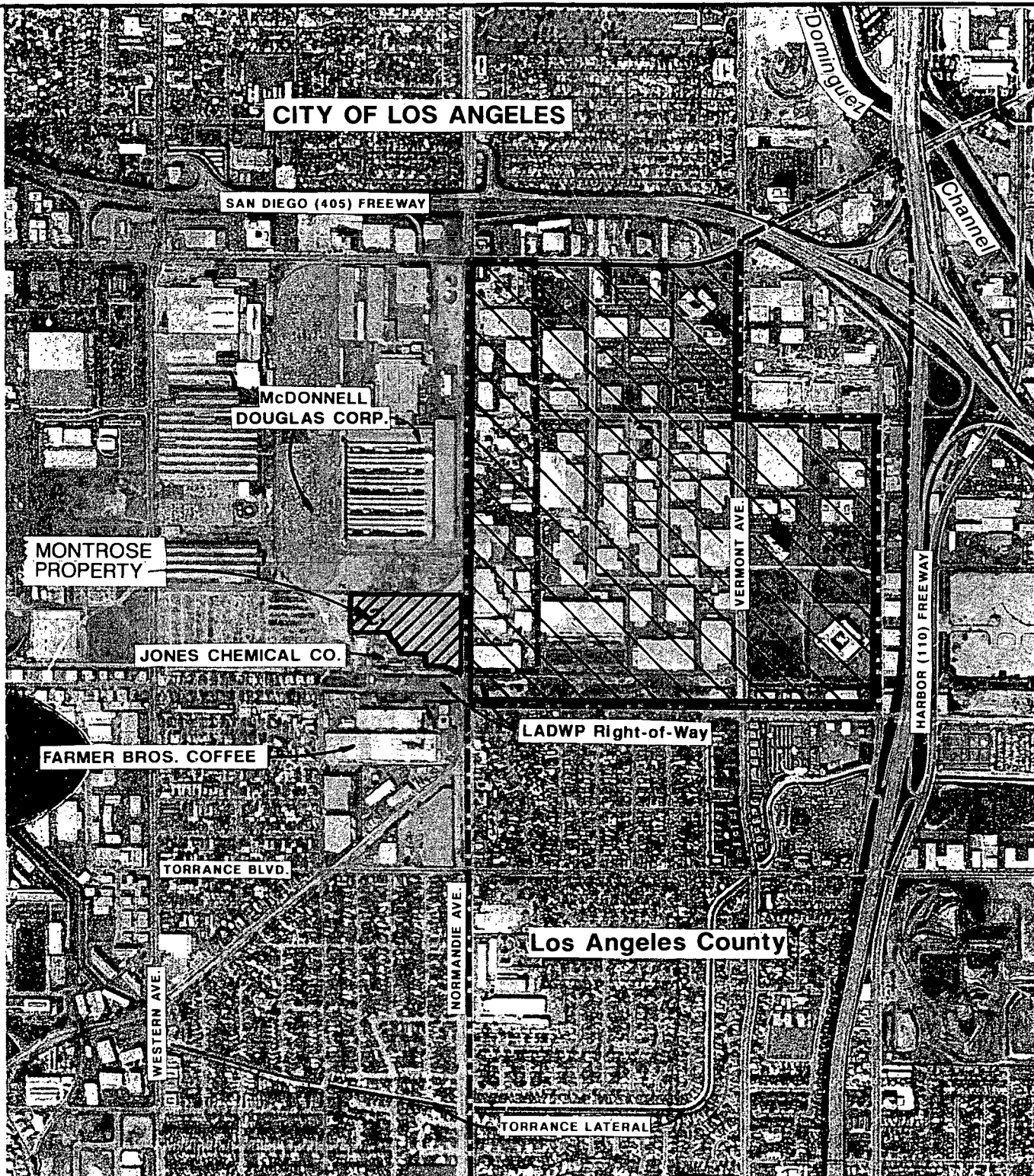


FIGURE 2-2  
AERIAL PHOTO OF THE  
MONTROSE STUDY AREA  
Ecological Risk Assessment  
Montrose Superfund Site



**LEGEND:**

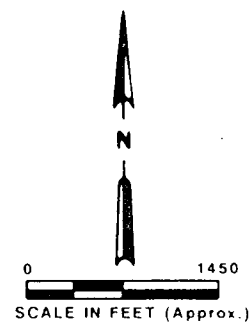


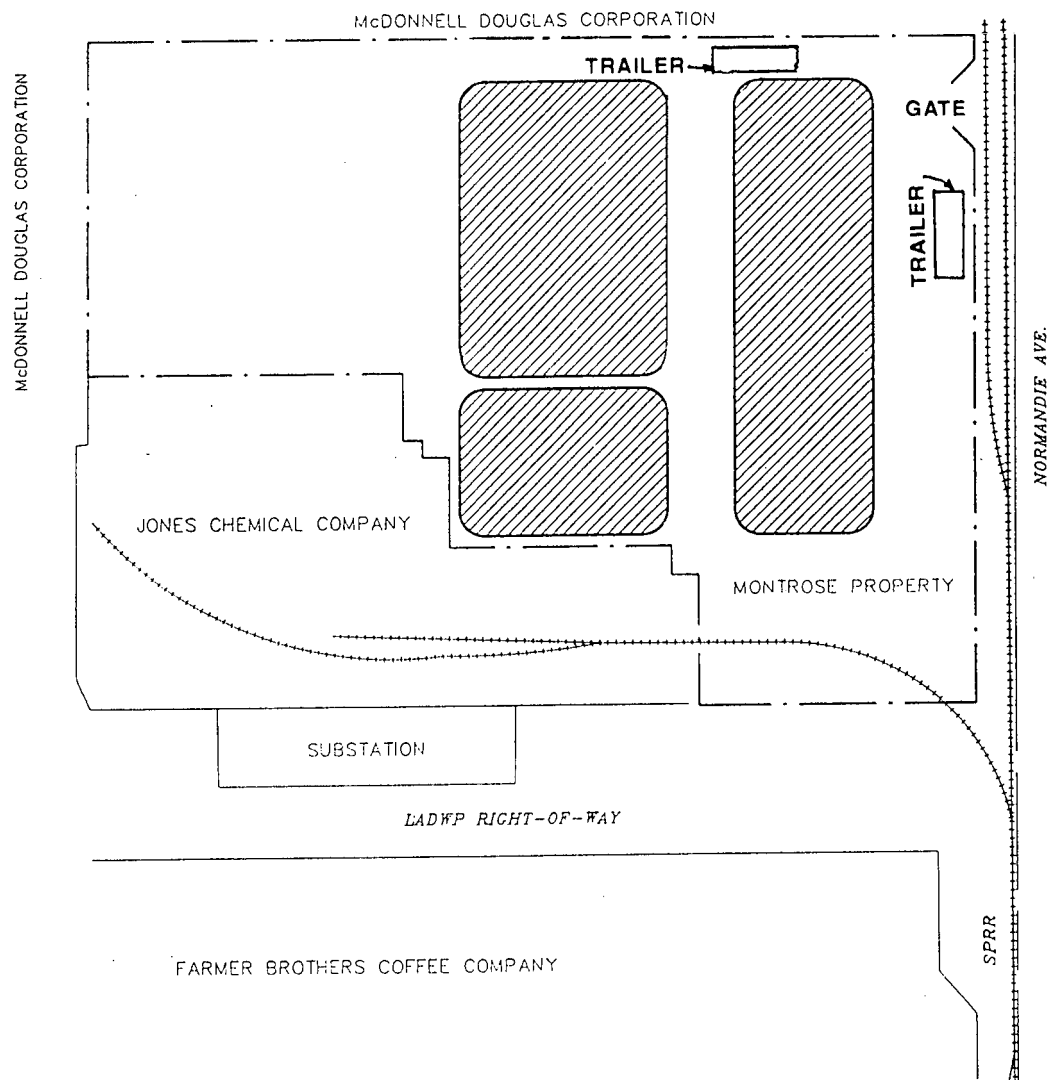
MONTROSE  
CHEMICAL CORP.








DEL AMO-Superfund Site

FIGURE 2-3  
LOCAL SETTING





## EXPLANATION

-  TOPOGRAPHIC HIGHS
-  MONTROSE PROPERTY BOUNDARY
-  RAILROAD TRACKS
-  SOUTHERN PACIFIC RAILROAD
-  LOS ANGELES DEPARTMENT OF WATER AND POWER

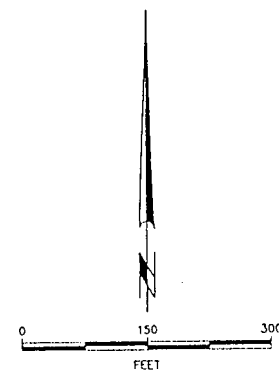


FIGURE 2-4  
CURRENT PROPERTY LAYOUT  
Ecological Risk Assessment  
Montrose Superfund Site

SOURCE: Hargis + Associates, 1990

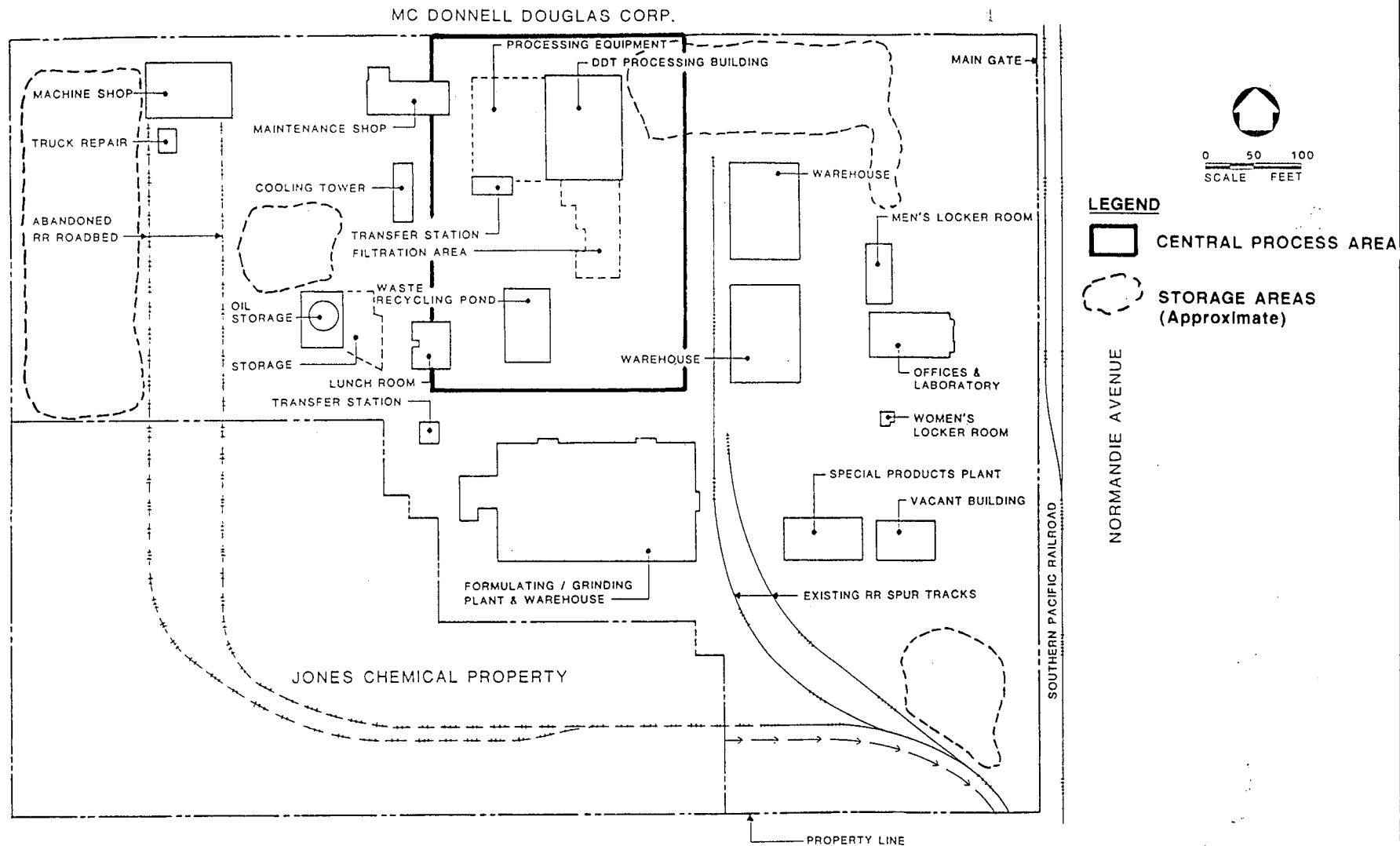


FIGURE 2-5  
HISTORICAL PROPERTY LAYOUT  
Ecological Risk Assessment  
Montrose Superfund Site

Source: Metcalf & Eddy, 1986

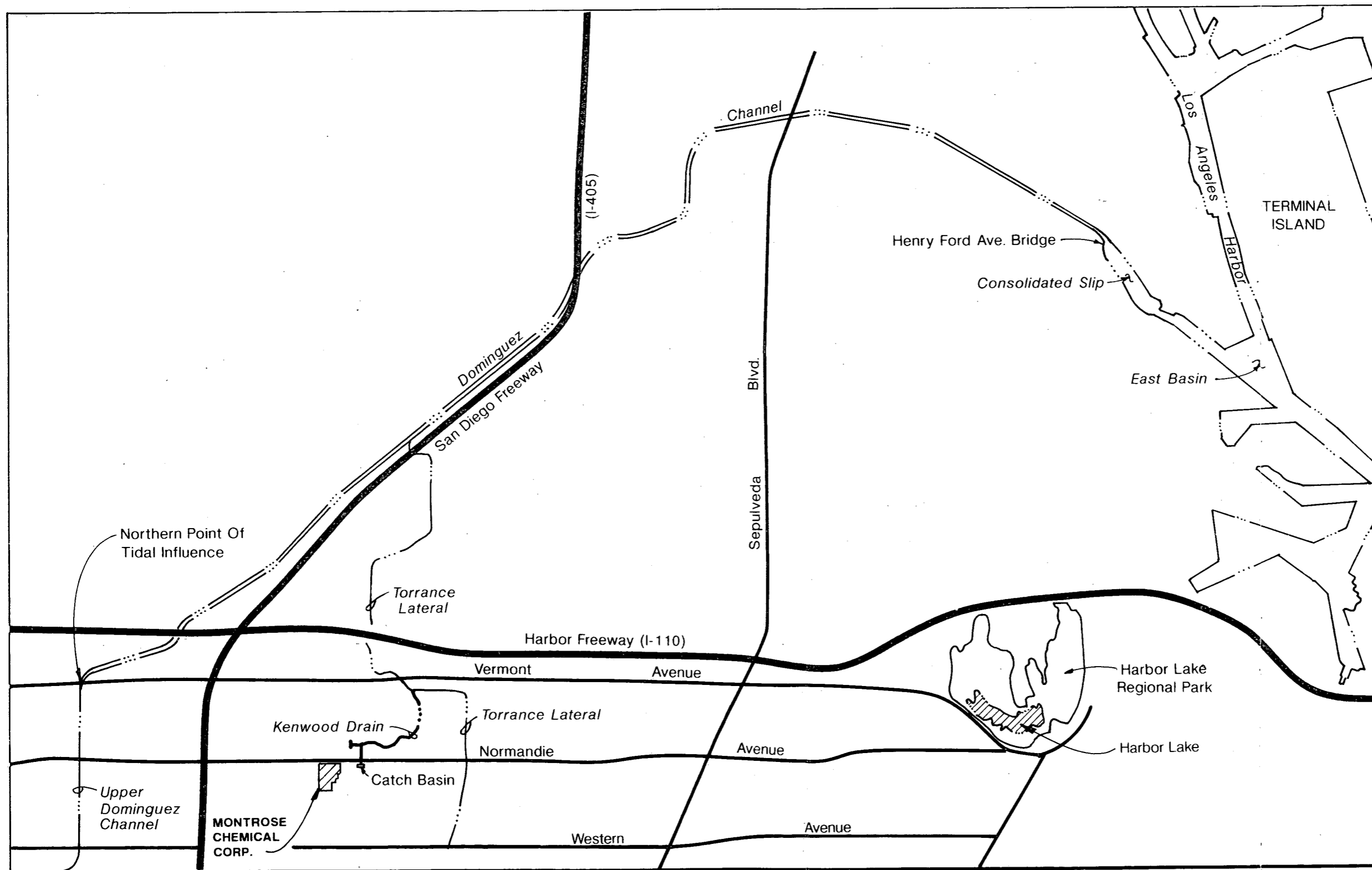
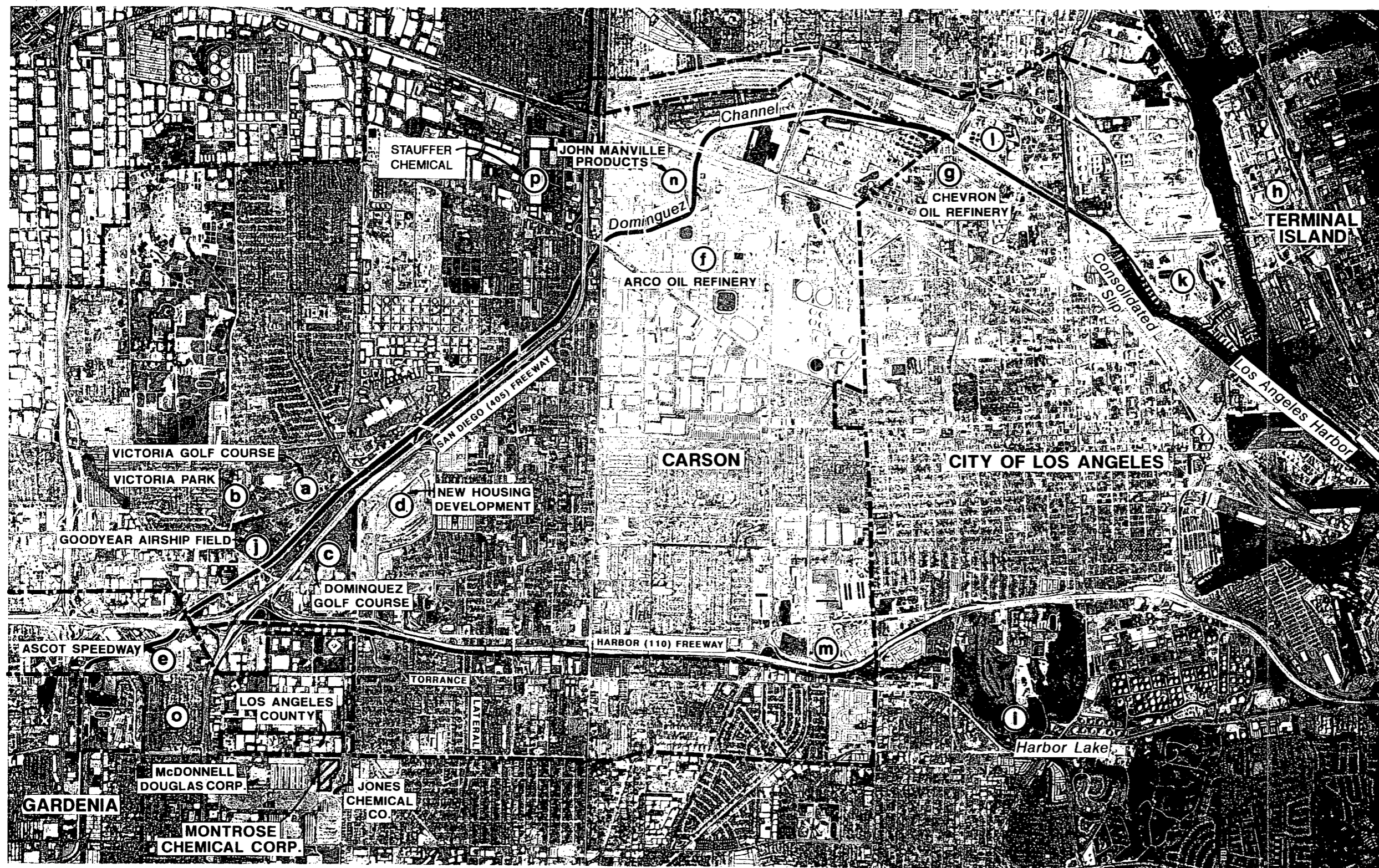


FIGURE 2-6  
FLOOD CONTROL CHANNELS  
SERVING THE  
MONTROSE AREA  
Ecological Risk Assessment  
Montrose Superfund Site

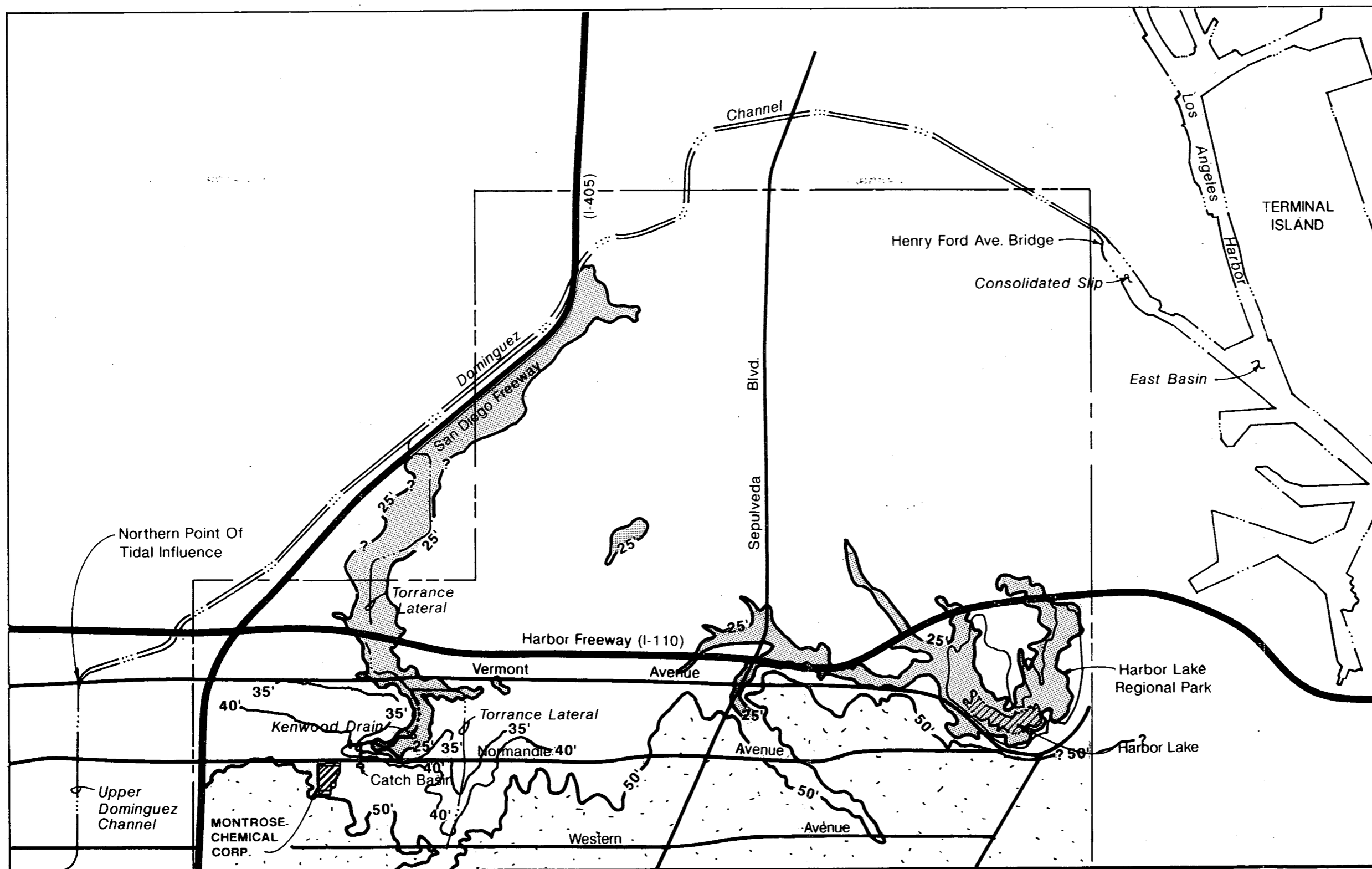


**LEGEND**

- (a) Victoria Golf Course
- (b) Victoria Park
- (c) Dominguez Golf Course
- (d) New Housing Development
- (e) Ascot Speedway
- (f) Arco Oil Refinery
- (g) Chevron Oil Refinery
- (h) Terminal Island
- (i) Harbor Lake
- (j) Goodyear Airship Field
- (k) Champlin Petroleum Co.
- (l) Texaco Inc.
- (m) Joint Water Pollution Control Plant (JWPCP)
- (n) John Manville Products
- (o) Cemetery
- (p) Stauffer Chemical Dominguez Facility
- City Boundary



FIGURE 2-7  
REGIONAL LAND USE  
Ecological Risk Assessment  
Montrose Superfund Site



**Legend:**

25'  
Contour in Feet above Mean Sea Level

= areas <25' msl

= areas >50' msl

area covered by Historical Maps

0 3600  
SCALE IN FEET (Approx.)

**FIGURE 2-8**  
**HISTORICAL TOPOGRAPHY DRAINAGE**  
**CONTOURS FOR AREAS POTENTIALLY**  
**AFFECTED BY PROPERTY RUNOFF**

Ecological Risk Assessment  
Montrose Superfund Site

SOURCE: City of Los Angeles, Public Works Drainage Maps: 599, 604, 605, 613, 614, dated 1958.

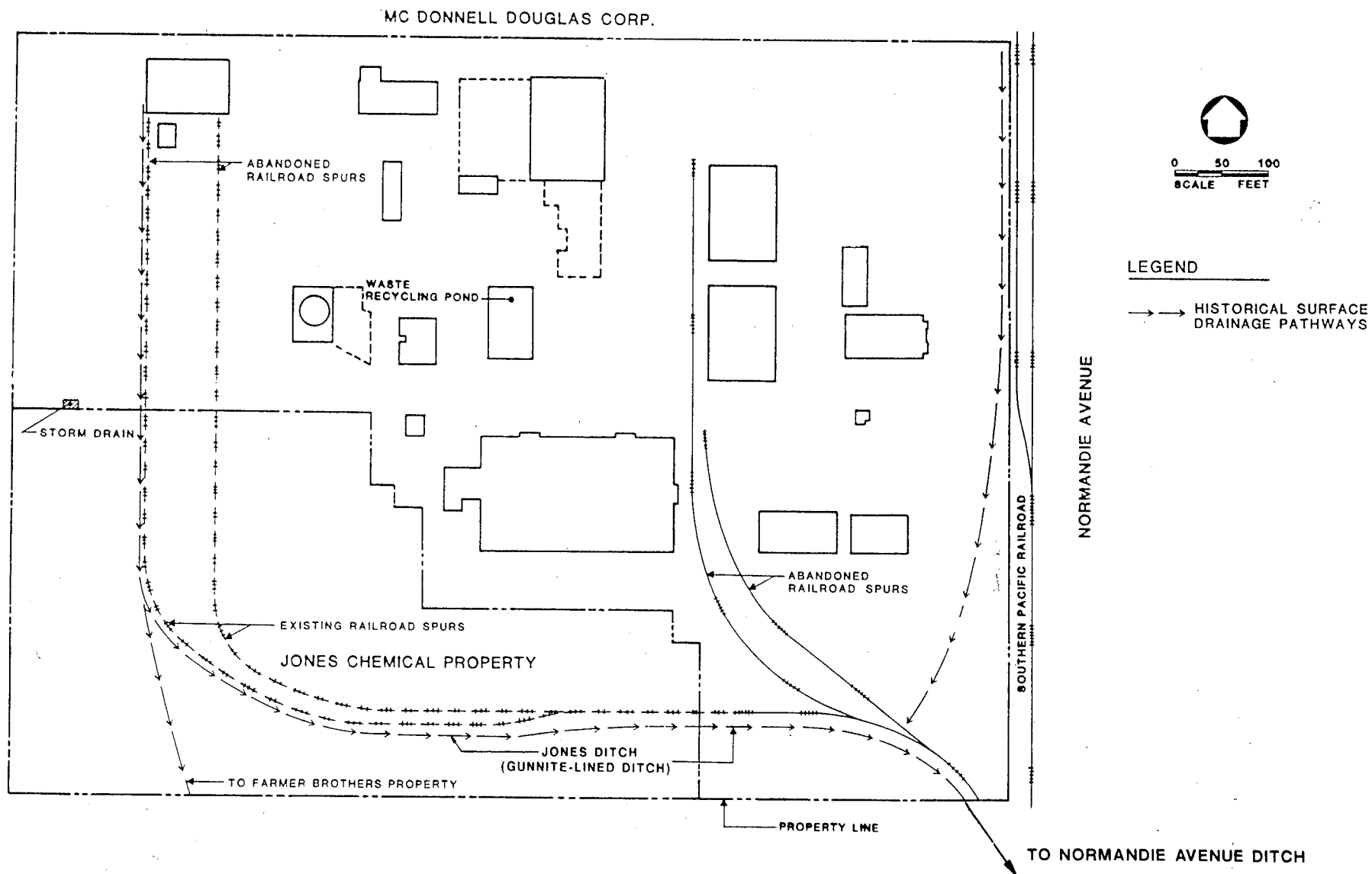


FIGURE 2-9  
HISTORICAL SURFACE WATER RUNOFF PATHWAYS  
Ecological Risk Assessment  
Montrose Superfund Site

Source: Metcalf & Eddy, 1986



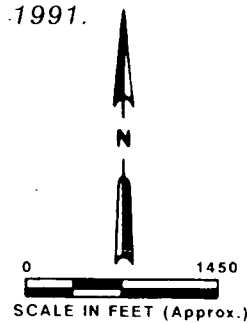
**LEGEND**

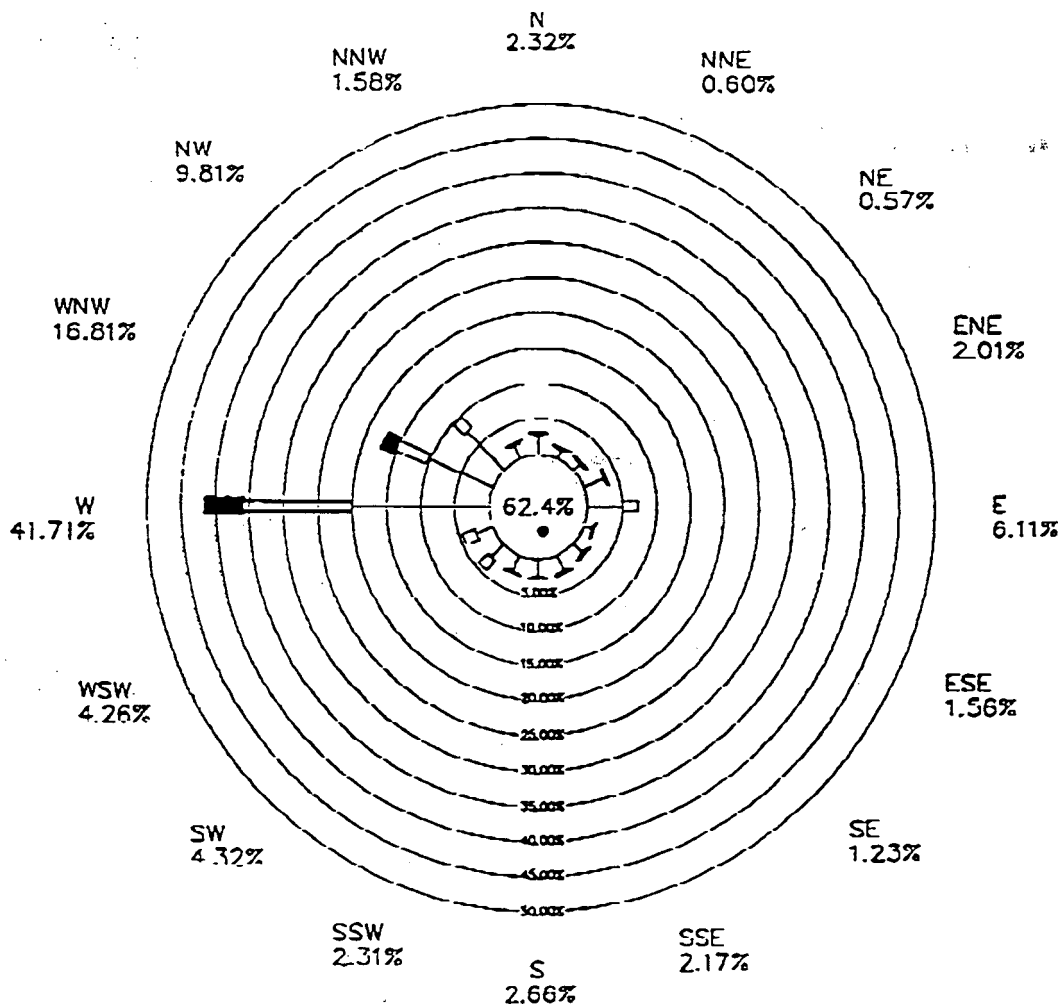
- STORMWATER DRAINAGE SYSTEM
- - - NORMANDIE AVENUE DITCH
- CATCHBASINS

SOURCE: LACDWP, 1991.

FIGURE 2-10  
DRAINAGE SYSTEMS  
CURRENTLY SERVING  
THE MONTROSE AREA

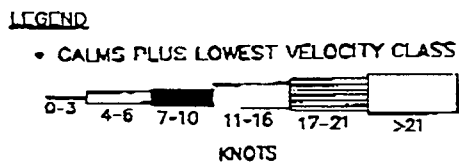
Ecological Risk Assessment  
Montrose Superfund Site





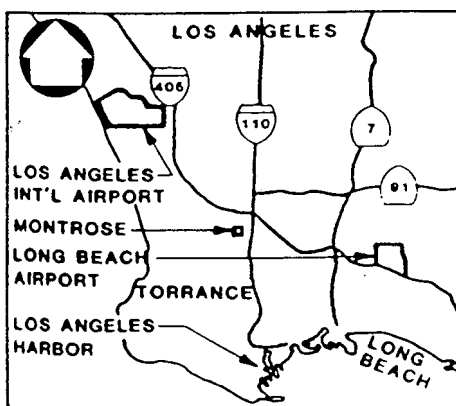
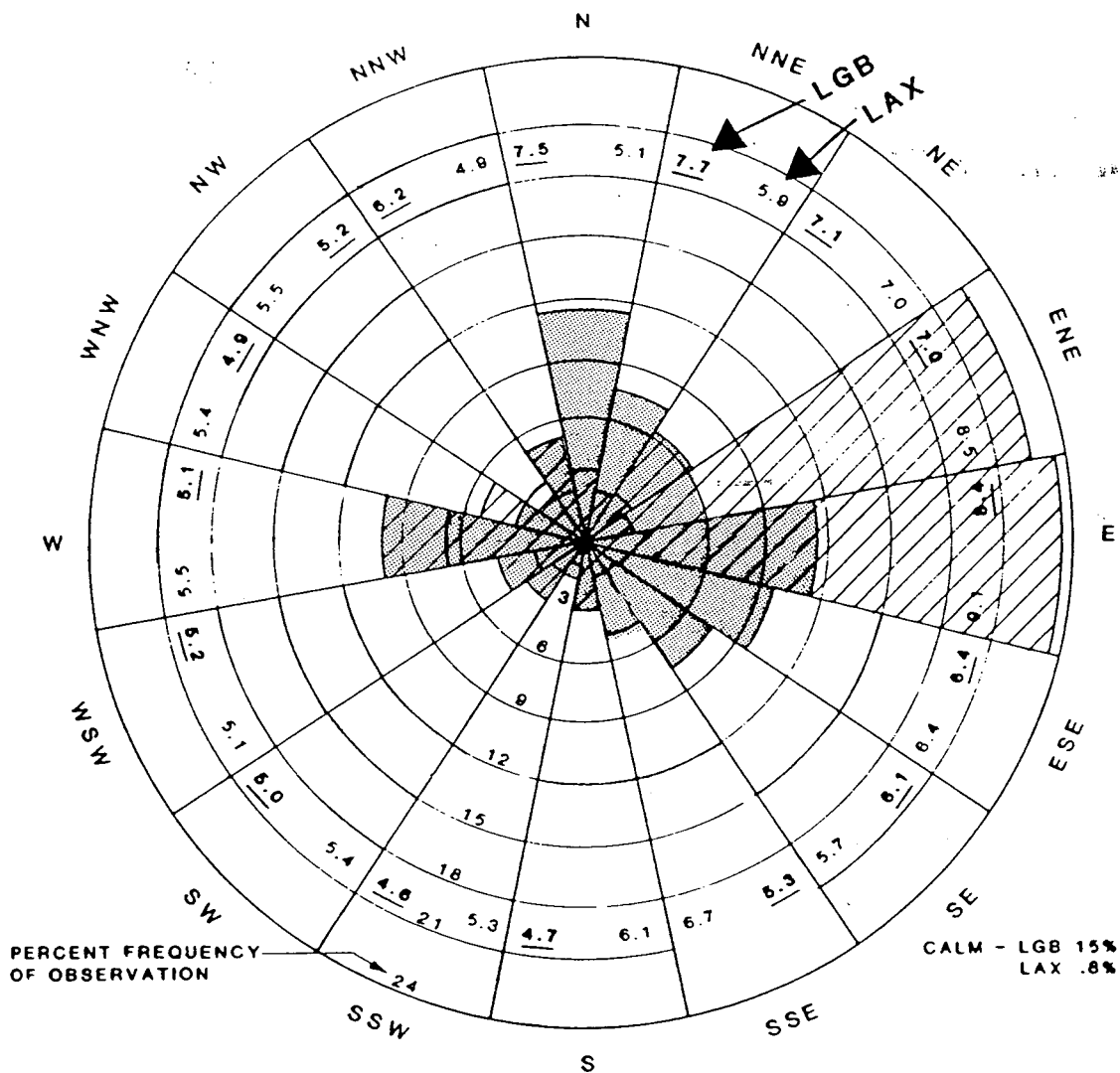
HOURLY AVERAGE SURFACE WINDS,  
PERCENTAGE FREQUENCY OF OCCURRENCE

STABILITY CLASS: ALL



SOURCE: SCAQMD, 1991.

FIGURE 2-11  
WIND ROSE FOR LENNOX, CALIFORNIA (1981)  
Ecological Risk Assessment  
Montrose Superfund Site



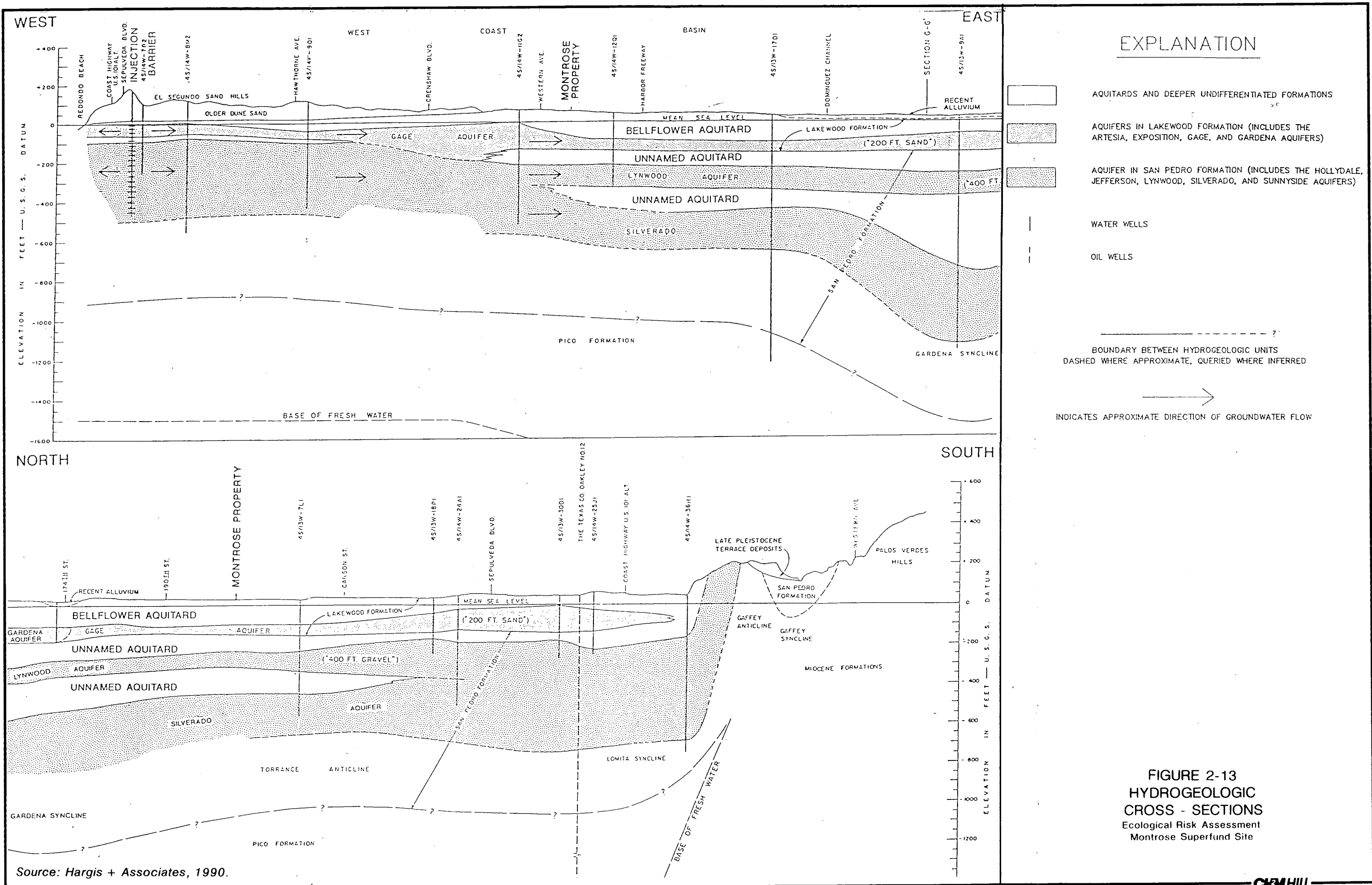
WIND ROSE - LEEWARD DIRECTION OF THE WINDS AT LOS ANGELES & LONG BEACH AIRPORTS. MEASURED AS PERCENT FREQUENCY OF OBSERVATION AVERAGED ANNUALLY FOR THE PERIOD 1965-74.

#### LEGEND

- LOS ANGELES AIRPORT (LAX)
- LONG BEACH AIRPORT (LGB)
- 6.4 AVERAGE WIND SPEED - LAX
- 6.4 AVERAGE WIND SPEED - LGB

SOURCE: Ecology & Environment, Inc., 1986.

FIGURE 2-12  
LEEWARD WIND ROSE FOR  
LONG BEACH AND LOS ANGELES AIRPORTS (1965-1974)  
Ecological Risk Assessment  
Montrose Superfund Site



Source: Hargis + Associates, 1990.

FIGURE 2-13  
HYDROGEOLOGIC  
CROSS - SECTIONS  
Ecological Risk Assessment  
Montrose Superfund Site

DRAFT

### **3 Nature and Extent of Contamination**

### Section 3

## Nature and Extent of Contamination

During its years of operation, Montrose released chemicals associated with its operations to the surrounding environment via:

- Discharge to sanitary sewers
- Disposal of wastes to the open ocean
- Hauling wastes to landfills
- Offsite transport of contaminated soils, surface water, and air
- Releases to groundwater

Compounds released likely included those found in the product (technical grade DDT), raw materials, and by-products associated with the production process. Investigations addressing the presence of these chemicals on the Montrose property and in surrounding areas are summarized below. The presence of chemicals detected in soil from these investigations is summarized in Table 3-1. Heavy metals were not among the ingredients known to be used in the operations, so they are not considered in this preliminary assessment.

### 3.1 Previous Onsite Investigations

The Montrose property initially came under the enforcement of the EPA and the Los Angeles RWQCB in November of 1982, following an EPA investigation. Elevated levels of DDT were detected in surface water runoff and sediments leaving the property.

**Table 3-1**  
**Organic Chemicals Detected in Soil from**  
**Various Investigations of the Montrose Chemical Plant**

Chemical	Central Process Area	Other On-Property Areas	Perimeter Areas <sup>b</sup>	LADWP Right-of-Way	Normandie Ditch <sup>a</sup>	Farmer Brothers Coffee	Neighborhood <sup>b,c</sup>
DDT	X	X	X	X	X	X	X
DDE	X	X	X	X	X	X	X
DDD	X	X	X	X	X	X	X
alpha-BHC	X	X	X		X		
beta-BHC	X	X	X	X	X	X	X
delta-BHC	X	X	X	X	X		X
gamma-BHC	X	X	X	X	X	X	X
Acetone	X	X	X	X			X
Carbon tetrachloride	X						
Chlorobenzene	X	X	X		X		
Chloroform	X	X	X				
Chlorophenols	X						
Dichlorobenzene isomers	X	X					
1,2-DCA	X						
1,2-DCE	X						
Ethylbenzene	X						
Hexachlorobenzene	X						
Hexachloroethane	X						
Methylene chloride	X	X		X			X
2-Butanone <sup>a</sup>	X	X					X
PCE	X	X	X	X			
Phthalates	X	X	X	X	X		X
PNAs			X				X
Styrene	X						
Toluene	X	X	X	X	X		X
1,2,4-Trichlorobenzene	X						
TCE	X		X				
Xylenes	X	X	X				

<sup>a</sup>2 butanone = methyl ethyl ketone

<sup>b</sup>Perimeter areas = Jones Chemical property, Southern Pacific Railroad (SPRR) right-of-way, McDonnell Douglas

<sup>c</sup>E&E, 1991

Source: H + A, 1990 unless otherwise noted.

The California Department of Health Services had collected "water residue samples" on February 26, 1981, from a drainage ditch (the Jones Ditch) serving both the Jones Chemical and Montrose properties and from the Normandie Avenue Ditch at the point where it discharged onto the Farmer Brothers Coffee Company property. Results of the sampling showed a pH of 14. DDT concentrations in the runoff ranged from 2.8  $\mu\text{g/L}$  to 98,000  $\mu\text{g/L}$ . Chlorobenzene was present at concentrations from 1,700  $\mu\text{g/L}$  to 84 percent (Montrose, 1981).

### **3.1.1 Montrose-Directed Activities**

Based on the results of the agency samples, Montrose was issued an enforcement order by the EPA and RWQCB to stop the releases of DDT, and to begin investigating possible soil contamination due to Montrose's operations by submitting plans for a remedial action (H + A, 1990). Montrose conducted sampling of subsurface soil from the Jones Ditch and the ponding area previously sampled by the Department of Health Services. A series of 37 soil samples were collected from 10 shallow borings (zero to 4 feet below ground surface [bgs]). As part of an unapproved remedial effort, Montrose constructed an earthen berm during the summer of 1983 to prevent storm-water runoff from leaving the property. During construction of the berm, Montrose drilled additional borings on-property and off-property to the south. DDT was found in soil at concentrations as high as 95,000 mg/kg. In 1984, the EPA proposed that the Montrose "Site" (the property and surrounding areas) be considered for ranking and potential inclusion in the Superfund National Priority List (NPL).

Montrose continued to conduct soil and groundwater investigations and take remedial actions independent of the EPA; in April 1985, Montrose graded and asphalt-capped the most of the property without the approval of the EPA. The capping was designed to control surface runoff and percolation. During the grading and capping, foundation pads for new buildings were made from redistributed shallow soil and fill. Grading involved reworking approximately the upper 3 feet of soil (M&E, 1986).

During April and May 1985, Montrose hired H + A to drill seven soil borings, one in the center of the former waste recycling pond and others throughout the property, and install and sample five wells in the upper Bellflower aquitard (Figure 3-1). All samples were analyzed for DDT, DDE, DDD, volatile organic compounds (VOCs) and chloral. These wells were sampled again in July 1985.

Twenty-one chemicals were detected more than once in the two rounds of groundwater sampling; analytical results are presented in Table 3-2. An additional 38 compounds were detected only once or were quantitated tentatively. Chemicals detected only once included: aldrin, delta-BHC, 1,1-DCA, trans-1,2-DCE, 2,4-dichlorophenol, bis(2-ethyl-hexyl)phthalate, naphthalene, 1,1,1-TCA, toluene, and phenol. Tentatively identified compounds (TICs) included: cyclic and substituted alkanes (e.g., cyclohexane), chlorinated sulfonyl/sulfone compounds, and chlorinated aromatic compounds (M&E, 1986). Many of the TICs are potential by-products of the DDT formulation process that used chlorobenzene and sulfuric acid.

Several patterns correlating analytical data results to historical Montrose activities (Figure 3-1) were noted:

- DDT, DDE, and DDD were primarily detected in groundwater from Well MW-2, which correlates with the waste recycling pond location. DDT was detected once (July 1985) in Well MW-1, southeast (downgradient) of the waste recycling pond, and once in Well MW-4 upgradient of the pond.
- The BHC isomers and aldrin were detected only in groundwater from Well MW-1, located just downgradient of the former formulating and grinding area.
- Benzene was detected in four of the five on-property wells, with the highest concentration found in Well MW-1 (3,200  $\mu\text{g/L}$ ).

**Table 3-2**  
**Chemicals Detected<sup>a</sup> in Groundwater Wells<sup>b</sup>**  
**(April, May and July, 1985)**  
**from H + A Sampling**

Chemical ( $\mu\text{g/L}$ )	MW-1 ( $\mu\text{g/L}$ )	MW-2 ( $\mu\text{g/L}$ )	MW-3 ( $\mu\text{g/L}$ )	MW-4 ( $\mu\text{g/L}$ )	MW-5 ( $\mu\text{g/L}$ )
DDT	- 17	630 2,400	-	- 36	-
DDE	- -	17 45	-	- -	-
DDD	- -	87 360	-	- -	-
alpha-BHC	200 220	- -	-	- -	-
beta-BHC	18 29	- -	-	- -	-
gamma-BHC (lindane)	20 33	- -	-	- -	-
delta-BHC	- 6.6	- -	-	- -	-
Benzene	660 3,200	- 150	40	- -	1,100
Carbon Tetrachloride	14 -	-	16	10 25	180
Chlorobenzene	1,400 15,000	54,000 180,000	59	850 160	93,000
Chloroform	1,100 1,600	5,800 5,600	760	3,100 4,700	24,000
2-Chlorophenol	31 -	30 37	-	- -	71
1,2-Dichlorobenzene	- 13	- -	-	- -	10
1,4-Dichlorobenzene	16 38	17 67	-	- -	27
1,2-DCA	3 -	150 150	-	- -	-
1,1-DCE	3 -	100 200	-	- -	-
Ethylbenzene	38 490	- -	-	- -	50
Methylene Chloride	63 120	- 400	-	- -	-
PCE	610 950	- -	14	1,100 1,300	580
1,2,4-Trichlorobenzene	11 31	- -	-	- -	10
TCE	30 -	- -	12	- -	25

<sup>a</sup>Chemicals detected only once are discussed in the text, but not presented in this table. For each chemical, top line is April/May sampling and bottom line is July sampling; dashes indicate nondetection of that chemical; blanks indicate the chemical was not an analyte in the analyses.

<sup>b</sup>All onsite (MW) wells have 10 feet of screening, between 61 and 77 feet bgs.  
Source: M&E, 1986

- Chloroform and chlorobenzene, detected in all on-property wells, were at their highest concentrations in Wells MW-5 (24,000  $\mu\text{g/L}$ ) and MW-2 (180,000  $\mu\text{g/L}$ ), respectively.
- Solvents, including ethylbenzene, methylene chloride, PCE, TCE, and several tentatively quantitated compounds (e.g., cyclohexane, cyclopentane, pentane, xylenes, butane) were detected in groundwater primarily from Wells MW-1 and MW-3. These wells are adjacent to the railroad spurs and downgradient from former maintenance and machine shops.

### 3.1.2 EPA Remedial Investigation, Part 1

Beginning in June 1985, the EPA and their contractor Metcalf and Eddy (M&E), conducted the field portion of a remedial investigation (RI) evaluating soil and groundwater from the Montrose property. During this work by M&E, referred to as the RI-Part 1, several chemicals were detected on a regular basis in soil and groundwater, including: DDT, DDE, DDD, chlorobenzene, dichlorobenzene isomers, benzene, carbon tetrachloride, tetrachloroethylene (PCE), trichloroethylene (TCE), chloroform, acetone, 2-butanone, and methylene chloride. BHC isomers were frequently detected in those samples it was analyzed for; however, samples were not consistently analyzed for BHC isomers. Two chemicals (2-butanone and methylene chloride) were considered to be potential laboratory contaminants (M&E, 1986).

M&E identified several potential source areas including: the waste recycling pond, the main production/processing area, storage and waste pile areas, grinding areas, and railway spurs (Figure 3-1). In addition, off-property sanitary sewers and storm drains were found to contain Montrose operations-related chemicals. M&E proposed that contaminant movement from the waste recycling pond into groundwater and soils was a continuing source of contaminant release off-property (M&E, 1986).

Based on the results of the RI-Part I, presented below, and the results of earlier Montrose-directed sampling efforts, the EPA was able to complete the hazardous ranking system process for inclusion of the Montrose property into the NPL (E&E, 1986).

### ***3.1.2.1 Soil Investigation***

To characterize soil contamination, 17 on-property and two off-property background soil borings were drilled during the RI-Part 1. Background surface soil samples were collected at the Van Ness Business Center, several blocks west of Western Avenue at Del Amo Boulevard, and in the Caltrans right-of-way on Normandie Avenue at the northeast corner of Artesia Boulevard (or 182nd Street, near Roosevelt Memorial Park; text and figures of M&E, 1986 disagreed). Chlorobenzene was the only chemical detected above 0.25 mg/kg in off-property samples; it was detected at the Normandie Avenue/Artesia Boulevard site at 8.4 mg/kg (surface sample) and at 1.9 mg/kg (deep sample) (M&E, 1986).

On-property soil boring locations are shown on Figure 3-2, and analytical results are tabulated in Table 3-3. Soil samples were analyzed for nine compounds: DDT, DDE, DDD, BHC, acetone, benzene, chlorobenzene, chloroform, and dichlorobenzene. Soil borings extended to a maximum depth of 9.5 feet bgs, with four exceptions: Boring 14D was sampled to 13.5 feet bgs, Boring 24D to 19.5 feet bgs, and Borings 25D and 46D were sampled to 11 feet bgs.

Shallow soil was found to contain DDT, DDE, and DDD in high concentrations across the property. In nine borings, DDT concentrations zero to 2 feet bgs exceeded 100 mg/kg. Only three borings had DDT concentrations below 100 mg/kg in the upper 2 feet bgs (Borings 34D, 35A, and 36D). Soil Boring 14D, located near the former processing and filtration areas, had the highest concentration of DDT (7,600 mg/kg, 1 to 1.5 feet bgs). Borings 14D and 24D (former waste recycling pond) contained DDT

**Table 3-3**  
**Analytical Results of Soil Samples (mg/kg)**  
**from M&E Sampling**  
**June 1985**

Chemical									
Depth (feet bgs)	DDT	DDE	DDD	BHC	Acetone	Benzene	Chlorobenzene	Chloroform	Dichlorobenzene
<b>11D</b>									
1.5-2	740	160	18				.44 J		0.25 J
3-3.5	80	1.3	2.9				.34 J		0.25 J
4-4.5	-	-	-	<0.001	0.45 J	<0.005	-	0.021 J	<0.001
4.5-5	-	-	-	<0.001	0.43 J	<0.005	-	0.02 J	<0.001
6-6.5	.067	.02 U	.04 U				.05 J		0.25 J
7.5-8	.04 U	.02 U	.04 U				.05 J		0.25 J
9-9.5	.016	-	-	<0.001	.045 J	<0.005	-	0.024 J	<0.001
<b>12D</b>									
1.5-2	270	60	9.8	0.25 J			.87 J		
3-3.5	310	23	4.7	0.25 J			.05 J		
4-4.5	-	-	-	-	.049 J	-	.1 J	0.02 J	
4.5-5	-	-	-	-	.043 J	-	.082 J	0.02 J	
6-6.5	.11 UJ	0.029 UJ	.04 U	0.25 J			.05 J		
7.5-8	.04 U	0.02 U	.04 U	0.25 J			.05 J		
8.5-9	-	-	-	0.25 J	0.024 J	-	.041 J	0.012 J	-
9-9.5	-	-	-	0.89 J	0.048 J	-	.078 J	0.02 J	-
<b>13D</b>									
1-1.5	1,600	650	35				.26 J		.34 J
1.5-2	1,200	230	280				.61 J		1.1 J
3-3.5	1,800	290	26		.099 J	-	1.9 J	.015 J	-
4-4.5	1,200	250	26				2.9 J		.73 J
4.5-5	1,400	340	36				.44 J		.25 J
5.5-6	.61	.079 UJ	.04 UJ				.05 J		.25 J
7.5-8	.1 UJ	.023 UJ	.04 UJ				.05 J		.25 J
9-9.5	-	-	-		.040 J	-	.04 J	.005 J	-
<b>14D</b>									
1-1.5	7,600	720	460				.45		<.25
2.5-3	1,200	700	160				2.3		<.25
3-3.5	2,100	270	260				3.0		<.25
4.5-5	1,300	170	67	<.001	4.9 J	<0.005	6.4	.68	<.001
6-6.5	1,600	67	43				910		<25
7.5-8	660	51	58				540		35
9-9.5	6,200	300	370	<.001	57	<0.005	7,100	72	<.001
10.5-11	11,000	170	910				14,000		<500
13-13.5	4,400	79	500				9,000		<500
<b>15D</b>									
1-1.5	1,100	40	72				4		<0.25
1.5-2	1,400	170	190				360		<0.25
3-3.5	2,600	260	72	<0.001	4.9 J	<0.005	15	0.29	<0.001
4-4.5	150	9.9	10				1.8		<0.25
7.5-8	320	0.81	1.2				0.49		0.44
9-9.5	-	-	-	<0.001	0.048 J	<0.005	0.02	0.018	<0.001

Continued

**Table 3-3**  
**Analytical Results of Soil Samples (mg/kg)**  
**from M&E Sampling**  
**June 1985**

Chemical									
Depth (feet bgs)	DDT	DDE	DDD	BHC	Acetone	Benzene	Chlorobenzene	Chloroform	Dichlorobenzene
<b>16D</b>									
1.5-2	1,100	2,200	110				0.083		<0.25
2.5-3	47	7	1.4				0.07		<0.25
3-3.5	<0.04	<0.02	<0.04				<0.05		<0.25
4.5-5	-	-	-	<0.001	0.043 J	<0.005	0.014	<0.005	<0.001
6-6.5	<0.04	<0.02	<0.04				<0.05		<0.25
7-7.5	<0.04	<0.02	<0.04				0.34		0.4
8.5-9	-	-	-	<0.001	0.049 J	<0.005	0.015	<0.005	<0.001
9-9.5	-	-	-	<0.001	0.012 J	<0.005	-	<0.005	<0.001
<b>21D</b>									
0-0.5	3,200	600	80				0.72 J		1.9 J
0.5-2	530	97	17				1.4 J		0.89 J
3-3.5	0.016	0.016	-	<0.001	0.03 J	<0.005		0.019 J	<0.001
5.5-6	0.04 U	0.02 U	0.04 U				0.05 J		0.25 J
6-6.5	0.04 U	0.02 U	0.04 U				0.05 J		0.25 J
7.5-8	0.04 U	0.02 U	0.04 U				0.05 J		0.25 J
9-9.5	-	-	-	<0.001	0.048 J	<0.005	-	<0.024 J	<0.001
<b>22D</b>									
1-1.5	670	140	21				0.43 J		0.25 J
1.5-2	1,804	50	5.9				0.57 J		0.53 J
2.5-3	0.04 U	0.02 U	0.04 U				0.05 J		0.25 J
3-3.5	-	-	-	<0.001	0.046 J	<0.005	-	0.02 J	<0.001
4.5-5	0.04 U	0.02 U	0.04 U				0.05 J		0.25 J
6-6.5	0.04 U	0.02 U	0.04 U				0.05 J		0.25 J
7.5-8	0.04 U	0.02 U	0.04 U				0.05 J		0.25 J
9-9.5	-	-	-	<0.001	0.036 J	<0.005	-	0.02 J	<0.001
<b>23D</b>									
1-1.5	1,200	220	51				1.6J		0.25J
1.5-2	1,700	210	70				1.1J		0.25J
3-3.6	0.021	0.016	--		--	--	0.1J	--	--
4-4.5	0.12UJ	0.034UJ	0.04U				0.05J		0.25J
6-6.5	0.086UJ	0.036UJ	0.04U				0.13J		0.25J
7.5-8	0.046	0.02U	0.04U				0.05J		0.54J
9-9.5	--	--	--		0.045J	--	0.14J	0.009J	--
<b>24D</b>									
1-1.5	880	200	52				1.1J		0.37J
1.5-2	2,000	550	100				7J		1.5J
2.5-3	1,200	120	53				7.1J		0.69J
4.5-5	3,800	530	130	<0.001	4.6J	<0.005	20J	<0.005	<0.001
5.5-6	44	7.4	2.1	0.401	0.037J	<0.005	0.16J	0.051J	0.66
6-6.5	0.04U	0.02U	0.04U				0.26J		0.25J
7.5-8	0.3	0.1	0.04U				2.7J		0.3J
9-9.5	4.7	4.7	0.23	42	0.097J	<0.005	1.1J	0.021J	4.66

Continued

**Table 3-3**  
**Analytical Results of Soil Samples (mg/kg)**  
**from M&E Sampling**  
**June 1985**

Chemical									
Depth (feet bgs)	DDT	DDE	DDD	BHC	Acetone	Benzene	Chlorobenzene	Chloroform	Dichlorobenzene
9.5-11	4,200	2,200	260				16,000J		370J
11.5-12.5	1,300	630	30	26	0.063J	<0.005	12,000J	<0.005	260
14-14.5	6,500	1,900	200				3,300J		65J
15.5-16	3,100	820	180				2,800J		66J
16-16.5	1,200	240	88				2,900J		42J
17.5-18	1,900	250	67	51	<0.1	<0.005	4,600J	<0.005	64
19-19.5	120	19	6				29J		2.2J
<b>25A</b>									
0-2	400	4.4	31				2.7		<0.25
2.5-3	<0.04	<0.02	<0.04				0.19		<0.25
3-3.5	<0.04	<0.02	<0.04				0.08		<0.25
4.5-5	0.007 J	-	-	<0.001	0.032 J	<0.005	0.006 J	0.052	<0.001
5.5-6	<0.04	<0.02	<0.04				0.08		<0.25
7.5-8	<0.04	<0.02	<0.04				0.05		<0.25
9-9.5	-	-	-	<0.001	0.048 J	<0.005		0.059	<0.001
<b>25D</b>									
0.5-1	110	11	8.4				0.08		<0.25
1.5-2	1,700	350	170				29		8.9
2-2.5	2,700	390	290				13		3.9
4-4.5	880	65	34	<0.001	0.062 J	<0.005	<0.014	<0.005	<0.001
5.5-6	<0.04	<0.02	<0.04				1.6		2.2
7-7.5	56	11	5.4				<0.05		<0.25
9-9.5	-	-	-	<0.001	0.061 J	<0.005	-	<0.005	<0.001
10.5-11	<0.04	<0.02	<0.04				<0.05		<0.025
<b>34D</b>									
1-1.5	0.98	0.44	0.04 U				3.2 J		0.25 J
1.5-2	7.1	1.1	0.43				3.7 J		0.25 J
3-3.5	580	57	35				0.31 J		0.45 J
4.5-5	630	94	17	27	0.048 J	0.005	0.83 J	0.005 J	<0.001
6-6.5	450	78	22				0.48 J		0.64 J
7-7.5	0.51	0.045	0.04 U				0.29 J		0.25 J
7.5-8	0.04 U	0.02 U	0.04 U				0.33 J		0.25 J
9-9.5	0.02	-	-	<0.001	0.047 J	0.005 J	0.032 J	0.029 J	<0.001
<b>35A</b>									
1-1.5	4.2	1.7	0.31				11 J		0.55 J
3-3.5	0.21	0.03	0.04 U				<0.05 J		0.25 J
4-4.5	0.04 U	0.02 U	0.04 U				<0.05 J		0.25 J
4.5-5	-	-	-	<0.001	0.05 J	0.005	0.046	0.015 J	<0.001
5.5-6	63	11	5.8				0.33		0.31 J
6-6.5	0.04 U	0.02 U	0.04 U				<0.05 J		0.25 J
7.5-8	0.042	0.02 U	0.04 U				<0.05 J		0.25 J
9-9.5	-	-	-	<0.001	0.059 J	0.005	0.11 J	0.023 J	<0.001

Continued

**Table 3-3**  
**Analytical Results of Soil Samples (mg/kg)**  
**from M&E Sampling**  
**June 1985**

Chemical									
Depth (feet bgs)	DDT	DDE	DDD	BHC	Acetone	Benzene	Chlorobenzene	Chloroform	Dichlorobenzene
<b>35D</b>									
1-1.5	1,600	320	160				1.1		2.0
1.5-2	1,400	170 J	34	15.1	0.032 J		0.018	0.014	<0.001
2-2.5	1,100	33	100				2.1		1.4
3-3.5	890	72	28	<0.001	5.9		22	0.5	<0.001
4-4.5	1,100	62	68				1.6		1.1
5.5-6	51	4.6	4				0.1		<0.25
6-6.5	10	1.1	1.7				<0.05		<0.25
9-9.5	0.38	0.029	-	0.012	0.042 J	<0.005	-	0.008	<0.001
<b>36D</b>									
0-2	0.92	0.19	0.063				<0.05		<0.25
3-3.5	<0.04	<0.02	<0.04				<0.05		<0.25
4-4.5	-	-	-	<0.001	0.037 J	<0.005		<0.005	<0.001
4.5-5	<0.04	<0.02	<0.04				<0.05		<0.25
6-6.5	<0.04	<0.02	<0.04				<0.05		<0.25
7.5-8	<0.04	<0.02	<0.04				<0.05		<0.25
8.5-9	0.007 J	-	-	<0.001	0.05 J	<0.005	-	<0.005	<0.001
9-9.5	-	-	-	<0.001	0.072 J	<0.005	-	<0.005	<0.001
<b>46D</b>									
1-1.5	22	2.5	1.4				<0.05		<0.25
1.5-2	190	33	15				0.12		<0.25
3-3.5	0.07	0.022	<0.04				<0.05		<0.25
4.5-5	0.051	0.018	-	0.09	0.031 J	<0.005	-	<0.005	<0.001
6-6.5	<0.04	<0.02	<0.04				<0.05		<0.25
6.5-7	0.44	0.075	<0.04				<0.05		<0.25
10-10.5	0.43	0.059		<0.001	0.035 J	<0.005	-	<0.005	<0.001
10.5-11	0.012 J	0.004 J	-	<0.001	0.043 J	<0.005	-	<0.005	<0.001
<b>Offsite Van Ness</b>									
Surface	0.17	0.011 J	0.03	<0.001	0.046 J	<0.005	0.15	<0.024	<0.001
Deep	0.033	-	-	<0.001	0.046 J	<0.005	-	<0.005	<0.001
<b>Offsite Artesia</b>									
Surface							8.4		<0.25
Deep							1.9		<0.25

**Notes:**

M&E did not define the following notations presented with the data; however, common definitions are presented.  
 J = data for limited use (qualitative only, not quantitative).  
 U = not detected above the limit of detection shown.

Source: M&E, 1986

at the greatest depths. The deepest sample collected in Boring 14D (13 to 13.5 feet bgs) contained DDT at 4,400 mg/kg. The two deepest soil samples from soil boring 24D contained 1,900 mg/kg of DDT (17.5 to 18 feet bgs) and 120 mg/kg (19 to 19.5 feet bgs).

Other chemicals were detected periodically at elevated concentrations. Of note are chlorobenzene concentrations detected in Boring 14D, with the highest concentrations at 10 feet bgs (14,000 mg/kg) and 13 feet bgs (9,000 mg/kg), and Boring 24D, where chlorobenzene was detected at 19 feet bgs. BHC, infrequently analyzed for in samples, had elevated concentrations detected in Boring 24D (9, 11.5, and 17.5 feet bgs), Boring 34D (4.5 feet bgs), and Boring 35D (1.5 feet bgs).

### **3.1.2.2 Groundwater**

The five existing Montrose groundwater wells were re-sampled by M&E. Analytical results of this sampling are presented in Table 3-4. Eight of the nine chemicals tested were detected in all five wells. Except for benzene and chloroform, the highest concentration of each compound was detected in groundwater from Well MW-2, just east of the former waste recycling pond. Chloroform was found at the highest concentration in Well MW-5 (22,000  $\mu\text{g/L}$ ) located in the northeast corner of the property, followed by Well MW-2 (5,900  $\mu\text{g/L}$ ). Benzene was detected at the highest concentration in Well MW-1 (5,000  $\mu\text{g/L}$ ) at the southeast corner of the property and in Wells MW-3 and MW-5. Benzene may be associated with historical activities from the Del Amo Superfund site.

BHC isomers, previously detected only in Well MW-1 during the H + A sampling, were detected in groundwater from all five on-property wells. Dichlorobenzene isomers were detected in two wells (MW-2 and MW-5) at higher concentrations than found by H + A, and were detected for the first time in Wells MW-3 and MW-4.

**Table 3-4**  
**Chemicals Detected in Groundwater**  
**from M&E Sampling**  
**June-August, 1985**

<b>Chemical (µg/L)</b>	<b>MW-1</b>	<b>MW-2</b>	<b>MW-3</b>	<b>MW-4</b>	<b>MW-5</b>
DDT	20	4,500	3	1.1	10
DDE	10	65 J	0.1	1	10
DDD	10	410	0.38	0.15 J	10
BHC isomers	185	330	1.54	2	178
Acetone	5,100	14,000	150 J	60 J	5,800
Benzene	5,000	0	80	0	1,700
Chlorobenzene	2,500	310,000	25	100	110,000
Chloroform	2,500	5,900	750	4,400	22,000
Dichlorobenzene isomers	123 J	736	60	60	180 J
Source: M&E, 1986					
J = for limited purposes only					

During this investigation, the RWQCB also collected groundwater samples; results are presented in Table 3-5. Again, results were similar to the H + A results, with a few exceptions. PCE was detected for the first time in Well MW-2 (750  $\mu\text{g/L}$ ). Chloroform was detected at 20,000  $\mu\text{g/L}$  in Well MW-4, a concentration four to five times higher than previously detected in that well.

M&E concluded that DDT, DDE, DDD, chlorobenzene, dichlorobenzene isomers, and acetone were present in groundwater most likely because of infiltration from the waste recycling pond. However, chloroform and benzene could be from on-property or off-property sources.

<b>Table 3-5</b> <b>Chemicals Detected in Groundwater from RWQCB Sampling</b> <b>July and August 1985</b>					
<b>Chemical (<math>\mu\text{g/L}</math>)</b>	<b>MW-1</b>	<b>MW-2</b>	<b>MW-3</b>	<b>MW-4</b>	<b>MW-5</b>
DDT (total)	24	60,600	na	52	na
Chlorobenzene	14,000	237,000	5	85	107,000
Benzene	4,000	190	710	na	1,900
PCE	980	750	23	990	500
Chloroform	2,400	9,300	630	20,000	20,000
Source: M&E, 1986 na = not analyzed					

### 3.1.3 EPA Remedial Investigation, Part 2

Beginning in October 1985, the remainder of the RI activities were conducted by H + A, under the terms of the Administrative Order on Consent (U.S. EPA Docket No. 85-04, as amended) reached between Montrose and the EPA (H + A, 1990). The

remainder of the RI (RI-Part 2) involved sampling groundwater, surface water, subsurface soil, and sediment in three phases:

- Phase 1      4/86 to 3/87    Soil and groundwater sampling on-property  
Soil, sediment, and surface water sampling off-property
- Phase 2A     8/88 to 5/89    Soil, groundwater, and sediment sampling on-property  
Soil, groundwater, and sediment sampling off-property
- Phase 2B     7/89 to 4/90    No onsite sampling  
Groundwater sampling off-property

In 1991, H + A drilled additional monitoring wells off-property to better define the plume in the Upper Bellflower Aquitard, Bellflower Sand, Gage Aquifer, and Lynwood Aquifer. Sampling of selected wells took place during 1991 and 1992, to monitor the downgradient extent of contamination.

### ***3.1.3.1 RI-Part II Soil Investigations***

Samples were collected from soil borings located both on- and off-property, and analyzed for pesticides (EPA method 608/8080) and VOCs (EPA methods 624-625/8240-8270). Organic chemicals detected in the central process area are presented in Table 3-6. Benzene was not detected in any soil samples.

<b>Table 3-6</b> <b>Volatile and Semivolatile Chemicals</b> <b>Detected in Central Process Area Soils from</b> <b>the RI-Part 2, H + A Sampling 1986 to 1990</b>		
DDT metabolites	Dichlorobenzene Isomers	PCE
BHC isomers	1,2-DCA	Phthalate
Acetone	1,2-DCE	Styrene
Benzoic Acid	Ethylbenzene	Toluene
Carbon Tetrachloride	Hexachlorobenzene	1,2,4-Trichlorobenzene
Chlorobenzene	Hexachloroethane	TCE
Chloroform	Methylene Chloride	Trichlorofluoromethane
Chlorophenols	Methyl ethyl ketone	Xylenes
Source: H + A, 1990		

A few borings consistently contained the highest concentration of chemicals; these borings correspond to the waste recycling pond (Borings S-101s and S-201), the area immediately downgradient of the pond (Borings S-202, S-203, and 24D), the area immediately east of the pond (Borings S-204 and MW-2s), and the DDT processing area (Borings S-304s and S-305s, and 14D).

DDT, DDE, DDD, and chlorobenzene were detected in soil to the depth of the water table, 70 feet bgs, in Boring 24D; DDT metabolites were detected in most of the soil samples analyzed. DDT was also detected in 22 of 25 soil samples collected from below the water table, 70 to 130 feet bgs. BHC isomers were detected above the water table in 30 percent of samples analyzed, but not from soil samples collected below the water table. The alpha- and beta-isomers of BHC were detected with 29 and 21 percent frequency, respectively; the other isomers were detected with less than 10 percent frequency. Chlorobenzene was detected in all soil borings from the central process area. Borings 24D (waste recycling pond), and S-304 and S-305 (DDT processing equipment area) had the highest concentrations of chlorobenzene above the water

table. Samples collected below the water table, including from both the upper and lower Bellflower Aquitards and the Bellflower Sands, also contained chlorobenzene. Movement of hydrophobic (water insoluble) chemicals (such as DDT) to groundwater may be from cosolvent effects of other chemicals (such as acetone) present in the soil and in groundwater.

On-property, but outside the central process area, DDT metabolites were detected at highest concentrations in shallow soil (0 to 6 feet bgs). The maximum depth at which DDT was detected was 11 feet bgs, except in boring MW-3 (west side of the property) where DDT was detected at 56 and 67 feet bgs. Most shallow soil samples had a combined DDT, DDE, and DDD concentration greater than 1,000 mg/kg. Other chemicals, including BHC, chloroform, dichlorobenzene, toluene, 2-butanone, PCE, 1,1,1-trichloroethane (1,1,1-TCA), TCE, and xylenes, were detected inconsistently in soil.

Surface soils from off-property areas were also collected. Samples taken from the Jones Chemical property, the Southern Pacific Railroad right-of-way, and the McDonnell-Douglas property contained several chemicals at greater than 5 percent frequency, including: the DDT metabolites, all BHC isomers, acetone, chlorobenzene, chloroform, PCE, phthalates, PCB-1260, toluene, TCE, xylenes, and several polynuclear aromatic hydrocarbons. DDT, DDE, and DDD were detected in 55 of the 56 samples collected. Samples collected from the LADWP right-of-way contained the same chemicals with a few exceptions: methylene chloride was detected, but chloroform, chlorobenzene, TCE, and xylenes were not. The LADWP right-of-way has been capped since these samples were collected. Samples collected from the Farmer Brothers Coffee Company property included the DDT metabolites and beta- and gamma-BHC.

### **3.1.3.2 Groundwater**

During the RI-Part 2, additional wells were installed into the major hydrogeologic units beneath the property (Table 3-7).

<b>Table 3-7</b> <b>Groundwater Wells Installed During H + A RI Activities, 1986 to 1990</b>		
<b>Hydrogeologic Unit</b>	<b>Direction of Groundwater Flow</b>	<b>No. of Wells</b>
Upper Bellflower Aquitard (72 to 85 ft bgs)	Southeast	26
Bellflower Sand (114 to 138 ft bgs)	East-southeast	17
Gage Aquifer (165 to 218 ft bgs)	East-southeast	14
Lynwood Aquifer (250 to 262 ft bgs)		3
Source: H + A, 1990		

Quarterly and semi-annual sampling was conducted; samples were analyzed for pesticides and VOCs. The following were detected:

- DDT was detected in groundwater samples primarily from wells located near the central process area; H + A reported that DDT, DDE, and DDD concentrations in groundwater decreased with distance from the central process area.
- BHC isomers were detected in the upper Bellflower Aquitard near the southeastern corner of the property and in two off-property wells screened in the Bellflower Sand.
- Chlorobenzene was the most widespread chemical detected in groundwater. It was detected in the upper Bellflower Aquitard to 180,000  $\mu\text{g/L}$ , in the Bellflower Sand to 45,000  $\mu\text{g/L}$ , and in the Gage Aquifer to 14,000  $\mu\text{g/L}$ .

- Benzene was detected in groundwater from the upper Bellflower Aquitard east of the property (near the Del Amo Superfund site), southwest of the property and the Jones Chemical Company, and south-southeast of the property. Benzene was also detected in the Bellflower Sand and the Gage Aquifer southeast of Montrose.
- Chloroform was detected at a maximum concentration of 74,000  $\mu\text{g/L}$  in the upper Bellflower Aquitard beneath the eastern portion of the property and the southeastern portion of the McDonnell-Douglas property. Chloroform was detected once in the Bellflower Sand but has not been positively detected in the Gage or Lynwood Aquifers.
- PCE, TCE, and 1,2-DCA were detected in the upper Bellflower Aquitard. PCE was detected in groundwater samples from wells on-property and to the south, beyond the Jones Chemical property.

In November of 1987, dense nonaqueous phase liquids (DNAPLs) were detected in the upper Bellflower Aquitard beneath the central process area. The DNAPLs contained chlorobenzene and DDT. H + A estimated that the DNAPL plume extended horizontally "several hundred feet east and north from the central process area." Well MW-2 was not sampled again following this discovery; however, the presence and rate of accumulation of free product were regularly monitored (H + A, 1990).

Light nonaqueous phase liquids (LNAPLs) were detected in two off-property wells, MW-7 and MW-20. LNAPLs in MW-7, south of the property, contained fuel hydrocarbons. LNAPLs in MW-20, northeast of the property, contained benzene.

Selected downgradient and on-property wells were sampled in July of 1992 to monitor migration of contaminants (H + A, 1992). A sample from Upper Bellflower Aquitard Monitoring Well MW-1 (southeast corner of property) contained chlorobenzene (53,000  $\mu\text{g/L}$ ), chloroform (18,000  $\mu\text{g/L}$ ), benzene (8,300  $\mu\text{g/L}$ ), and PCE (3,400  $\mu\text{g/L}$ ).

In the Bellflower Sand, chlorobenzene was detected 0.6 mile south of the Montrose property (Monitoring Well BF-31; 370  $\mu\text{g/L}$ ) and over 1 mile southeast of the property (BF-26; 20  $\mu\text{g/L}$ ). Chlorobenzene was detected in the Gage Aquifer 0.5 mile southeast of the Montrose property in Monitoring Well G-19 (180  $\mu\text{g/L}$ ), and in Monitoring Well G-3 (490  $\mu\text{g/L}$ ) at the south edge of the property. Chlorobenzene was also detected in the Lynwood Aquifer. The sample collected from Monitoring Well LW-1, on-property, had a concentration of 470  $\mu\text{g/L}$ .

## **3.2 Previous Off-Property Investigations**

### **3.2.1 Surface Water Runoff Pathways**

Several studies attempted to examine the chemical content of water and/or sediments from drainages serving the area. Some of these included off-property RI activities, sediment sampling for proposed dredging activities, and routine sampling of surface water by various agencies.

#### **3.2.1.1 RI Activities**

As part of the Montrose RI-Part 2, in 1986 and 1988, H + A sampled sediment and surface water from the Kenwood Drain, Torrance Lateral, Dominguez Channel, and Consolidated Slip. H + A measured the depth of sediments and collected samples for analysis. Sediment samples were also collected in 1988 from the Dominguez Channel. The U.S. EPA determined that the samples collected in 1986 and 1987 were not valid for any purpose. Only information on sediment accumulation patterns is presented for the 1986 field effort; however, both sediment and analytical results for the 1988 effort are presented. Overall sediments were found in intermittent deposits along the Kenwood Drain and Torrance Lateral. At the confluence of the Torrance Lateral and the Dominguez Channel, sediments as thick as 2 feet were observed in the Torrance

Lateral. The most sediments (5.6 feet thick) were found in the Dominguez Channel 200 feet downstream of the Torrance Lateral (H + A, 1990).

Analytical results from surface water samples were collected in 1986 and 1987, during wet and/or dry weather periods from the Normandie Avenue Ditch, Jones Ditch, Torrance Lateral, Dominguez Channel, and Consolidated Slip.

**Kenwood Drain.** Sediment depth was measured from the Kenwood Drain in 1986 (Figure 3-3). Five locations inspected along the Kenwood Drain contained sediments, which varied in thickness, but none were present at several other sites. Three locations had 1 to 4 inches of sediment deposits, including location KD-7 (where the Kenwood Drain feeds into the Torrance Lateral), which had a "sandbar" 4 inches deep and 1.5 feet wide. Sample location KD-4 had 7 inches of deposited sediments, and location KD-5 had areas with sediment depths of 8 to 18 inches.

**Torrance Lateral.** Sediment locations in the Torrance Lateral were characterized in June of 1986. There were five areas along the Lateral where sediments were found (Figure 3-4). Four of the five locations were described as sand bars ranging from 0.5 to 3 inches thick. A large sand bar approximately 2 feet thick was present at the confluence of the Lateral and the Dominguez Channel. H + A reported that in June 1987, "there was evidence that the large sediment deposit located near the confluence of the Lateral with the Dominguez Channel had been partially removed using heavy equipment" (H + A, 1990). This corresponds with invert cleaning of the Lateral in May of 1987.

**Dominguez Channel.** During the 1986 sampling, surface water depths in the Dominguez Channel ranged from 6.9 to 12 feet deep at the five SED sample locations (Figure 3-5). H + A noted during both wet and dry sampling events that the tidal current appeared to counter the downstream flow at the sample points downstream from the Torrance Lateral (SED-13, SED-14 and SED-15).

As a later phase of the RI (Part 2), H + A conducted a sediment survey in 1988 at 20 locations along 7,200 feet of the Dominguez Channel around the confluence with the Torrance Lateral (Figure 3-5) (H + A, 1990). Sediments were surveyed along transects that crossed the channel, with sediment depth measurement and sample collection near each side and in the center of the channel. Along the western side of the channel, sediment depth ranged from 0.1 to 5.6 feet thick; in the center, thickness ranged from 0 to 3.3 feet thick; and along the eastern bank, the sediments were from 0 to 2.6 feet thick. The deepest sediment deposits were from 800 feet upstream to 200 feet downstream of the confluence; over 60 percent of these locations had greater than 2 feet thickness of sediments. Several transects downstream from the confluence had little or no sediments present; the smallest amount of sediments was detected 3,000 to 5,000 feet downstream from the confluence.

Selected sediment samples from this 1988 field effort were analyzed for total DDT/DDE/DDD; results are presented on Figure 3-5. The highest concentration of DDT/DDE/DDD was detected in Sample T1C-0.5, with 4.1 mg/kg in sediments. Total organic carbon (TOC) was measured in only two samples (which did not include T1C-0.5).

**Consolidated Slip.** Ten sediment and surface water samples were collected from the Consolidated Slip during the RI-Part 2 study conducted in 1986. Water depths at these locations varied from 10 to 33 feet deep.

Before the RI, samples were collected in 1973 and 1978 from four areas of the Los Angeles Harbor, including the Consolidated Slip, to support a proposed dredging project. Sediment samples from the Consolidated Slip and the East Basin were analyzed for total DDT in 1973. Total DDT in sediments was reported at 0.0227 mg/kg in the Slip and at 0.0244 mg/kg in the East Basin samples (Clark, 1982).

Sampling in 1978 indicated that maximum DDT levels increased at the Los Angeles entry to the harbor, but they apparently declined elsewhere in the harbor (Soule and

Oguri, 1980). Although DDT had been high at the mouth of the Dominguez Channel in 1973, it was not detected there in 1978. DDT and its metabolites were the only chemicals of concern in this risk assessment that were measured in the 1973 and 1978 studies.

#### **3.2.1.2 E&E Sampling**

In June 1991, EPA contractor E&E conducted a Listing Site Inspection Summary Report for the Stauffer Chemical Dominguez Facility (Figure 2-7), a pesticide manufacturing facility in Carson, California. Montrose was a partially owned subsidiary of the Stauffer Chemical Company. From the 1940s through the 1960s, the Stauffer facility periodically stored, milled, and packaged pesticides for Montrose (E&E, 1991). As part of this study, five sediment samples were collected from Project 1202 (a flood control channel parallel to Wilmington Avenue) and four from the Dominguez Channel (Figure 3-6). Samples were analyzed for DDT, DDE, and DDD. Project 1202 channel sample results were consistently below quantitation limits or not detected (Table 3-8). However, sediment samples from the Dominguez Channel contained DDE and DDD at elevated levels. While DDT was positively detected in only one sample (SS-7), limits of detection were high. Samples were re-analyzed in October 1991 because of various data quality control issues; however, because of holding time constraints, the October 1991 data can be used only for qualitative purposes. Even with these constraints, DDT metabolites were detected at significantly higher concentrations in the Dominguez Channel than in Project 1202 sediments. The two closest sample locations downstream of the Torrance Lateral, SS-7 and SS-9, had the highest reported DDT levels.

#### **3.2.1.3 STORET Information**

The EPA STORET data base is a repository of water quality information obtained by state and federal agencies. This data base was searched in May of 1992 for information collected from sample stations within the Montrose study area. Available sample sta-

tions were identified in the Dominguez Channel and in the Torrance Lateral. Analytical results were obtained for total DDT/DDE/DDD, total BHC isomers, and chlorobenzene for the years 1977 through 1990; results were not available for other chemicals of interest. Results are discussed below.

**Table 3-8**  
**E&E Sediment Sample Results<sup>a</sup>**  
**Project 1202 and the Dominguez Channel**  
**June 22 and 23, 1991**

Chemical ( $\mu\text{g/kg}$ )	Project 1202 Samples					Dominguez Channel Samples			
	SS-1	SS-2	SS-3 <sup>c</sup>	SS-4	SS-5	SS-6	SS-7	SS-8	SS-9
DDT	3*	14*	7*	<40	<40	56*	84	35*	42*
	1*	0.6*	1*	0.4*	1*	22*	63*	31*	60*
DDE	2*	6*	4*	<40	3*	82	130	100	120
	1*	0.9	1	0.5*	1*	33*	64*	73*	80*
DDD	2*	7*	6*	<40	4*	91	150	110	150 <sup>b</sup>
	1*	1	1	0.4*	1*	38*	75*	89*	96*

<sup>a</sup> Results represent analysis in June 1991 (top) and re-analysis in October 1991 (bottom). Because of analytical and holding time considerations, the October results all have J qualifiers.

<sup>b</sup> Text and table in source document differed. Text stated 150  $\mu\text{g/kg}$ , table stated 140  $\mu\text{g/kg}$ .

<sup>c</sup> SS-3 is a duplicate of SS-2.

\* The data are qualitatively acceptable, but usable for limited purposes only.

Source: E&E, 1991

**Torrance Lateral.** Samples were collected in the Torrance Lateral at the Main Street overpass (Figure 3-7). Figure 3-8 illustrates the results of sampling. Chemicals associated with Montrose activities (DDT/DDE/DDD or BHCs) were detected only four times and at lower concentrations after December 1984; this corresponds with the capping of the Montrose property in April of 1985.

Samples were collected and analyzed regularly for DDT from 1977 through 1990 (Figure 3-8). When DDT was positively detected, the concentrations ranged from 0.02  $\mu\text{g/L}$  to 39.2  $\mu\text{g/L}$ , and limits of detection were below 0.2  $\mu\text{g/L}$  with the exception of July 22, 1986, (2.0  $\mu\text{g/L}$ ) and July 11, 1990 (0.5  $\mu\text{g/L}$ ). Of 77 samples where DDT was detected, 22 had concentrations exceeding 0.5  $\mu\text{g/L}$ ; 14 of those exceeded 1  $\mu\text{g/L}$ , and three exceeded 10  $\mu\text{g/L}$ . The three exceeding 10  $\mu\text{g/L}$  were collected on December 4, 1980 (12.91  $\mu\text{g/L}$ ), January 28, 1981 (39.2  $\mu\text{g/L}$ ), and March 2, 1981 (18.7  $\mu\text{g/L}$ ). DDT was detected twice after May of 1984, at 0.3  $\mu\text{g/L}$  (January 4, 1987) and at 0.25  $\mu\text{g/L}$  (December 5, 1990).

DDE data are available from 1977 through 1987, but DDE was not detected after May 1984. With only two exceptions, limits of detection were 0.2  $\mu\text{g/L}$  or lower. DDE was detected below 0.5  $\mu\text{g/L}$  for 60 out of 73 samples; it was detected between 0.5 and 5  $\mu\text{g/L}$  11 times. DDE exceeded 5  $\mu\text{g/L}$  only twice, on December 4, 1980 (5.13  $\mu\text{g/L}$ ) and on January 6, 1981 (7.48  $\mu\text{g/L}$ ). Periods of elevated DDE show a general correlation with periods of elevated DDT.

DDD data are also available from 1977 through 1987; however, only two samples (non-detects) were analyzed after 1984, in 1985 and 1987. DDD concentration was below 0.5  $\mu\text{g/L}$  for 53 out of 60 times it was detected. DDD concentrations exceeded 1  $\mu\text{g/L}$  only 4 times; it was detected at 1.38  $\mu\text{g/L}$  (January 9, 1980), 4.1  $\mu\text{g/L}$  (January 28, 1981), 1.47  $\mu\text{g/L}$  (March 2, 1981), and 1.87  $\mu\text{g/L}$  (October 7, 1981).

BHC isomers were also detected in water from the Torrance Lateral at Main Street. Gamma-BHC (lindane) results are available for 1977 through 1990; although analyzed for several times per year, it was detected only twice after 1984. Limits of detection were regularly below 0.1  $\mu\text{g/L}$  with three exceptions, which were all below 0.5  $\mu\text{g/L}$ . Ten samples contained BHC between 0.1 and 0.2  $\mu\text{g/L}$  and six samples contained BHC between 0.2 and 1.0  $\mu\text{g/L}$ . Lindane exceeded 1  $\mu\text{g/L}$  twice, on October 7, 1981 (1.41  $\mu\text{g/L}$ ) and on March 4, 1983 (2.05  $\mu\text{g/L}$ ). Beta-BHC was analyzed in samples collected between November 1988 and December 1990; it was never detected.

**Dominguez Channel.** Two STORET data base sampling areas were identified at the northern end of the Dominguez Channel (Figure 3-7): upstream of Vermont Avenue and below the Vermont Avenue Bridge (referred to in the STORET data base three separate ways: "below Vermont Avenue," "downstream of Vermont Avenue," and "Vermont Avenue Bridge"). Samples were also collected at the 190th Street overpass and at the Henry Ford Avenue Bridge. Samples were analyzed for DDT, DDE, DDD, and lindane beginning in 1973 and continuing through 1990.

Upstream of Vermont Avenue, DDT was positively detected 81 times; however, concentrations exceeded  $0.5 \mu\text{g/L}$  only four times, only one of those four times did it exceed  $1.0 \mu\text{g/L}$  (March 2, 1982, at  $1.29 \mu\text{g/L}$ ). Detection limits regularly were at or below  $0.1 \mu\text{g/L}$  for all but those four samples. DDE and DDD were detected periodically from 1976 and 1978, respectively, through April of 1984, but not after 1984. The maximum concentration of DDE detected was  $0.65 \mu\text{g/L}$ ; detected concentrations equalled or exceeded  $0.2 \mu\text{g/L}$  only 12 times. For DDD, the maximum concentration detected was  $0.215 \mu\text{g/L}$  (January 1980); only four samples exceeded  $0.1 \mu\text{g/L}$ . Lindane was detected at a maximum concentration of  $0.71 \mu\text{g/L}$  (November 1979). Over a 20-year period, it was detected 109 times; the concentration of  $0.1 \mu\text{g/L}$  was exceeded 30 times, of which nine times exceeded  $0.2 \mu\text{g/L}$ .

At the sample location just below Vermont Avenue, samples were analyzed infrequently for DDT metabolites or BHC isomers. DDT had a maximum detected concentration of  $0.49 \mu\text{g/L}$  (December 4, 1974); DDE was detected only four times, with a maximum concentration of  $0.13 \mu\text{g/L}$  (December 4, 1974), and DDD was detected only once at a concentration of  $0.3 \mu\text{g/L}$  (December 4, 1974). Lindane was detected seven times with the maximum of  $0.164 \mu\text{g/L}$  on November 12, 1976. These chemicals were last detected in this location in May of 1977; they have only been sampled from this location once since then, on October 15, 1986, when none were detected.

At the 190th Street overpass, three samples collected during late 1975 and early 1976 contained maximum concentrations of  $0.62 \mu\text{g/L}$  DDT,  $0.35 \mu\text{g/L}$  DDE,  $0.37 \mu\text{g/L}$

DDD, and 0.18  $\mu\text{g/L}$  lindane. Samples collected from the Henry Ford Avenue Bridge were analyzed only three times for DDT or BHC-related chemicals. DDT and metabolites were detected only once (February 3, 1975) at 0.23  $\mu\text{g/L}$  DDT, 0.03  $\mu\text{g/L}$  DDE, and 0.05  $\mu\text{g/L}$  DDD. Lindane was detected three times, most recently at 0.01  $\mu\text{g/L}$  (May 2, 1977).

### **3.2.2 Downwind Areas**

Activities at the Montrose property, during the years of operation, reportedly released DDT and other related chemicals to the atmosphere. Some of these activities included the formulation, grinding, and packaging operations (E&E, 1986). Montrose facilities included a grinding operation; particulates from this process were likely transported aerially to surrounding environments.

Demolition of the property buildings and activities associated with paving the property (e.g., grading) may have resulted in temporary increases in DDT emissions. Following the facility's closure in 1982 and its covering in 1985, emissions may have been reduced but no data are available to assess these changes.

#### **3.2.2.1 *Southern California Coastal Water Research Project Studies***

Offshore aerial fallout studies for DDT/DDE/DDD were conducted by the Southern California Coastal Water Research Project Authority (SCCWRPA) in the spring of 1973 and 1974 (Young et al., 1980). Monthly mass emission rates (kg/month) were calculated from 26 weekly aerial fallout measurements at 11 sites (Figure 3-9). Areas with the greatest mass of DDT/DDE/DDD in aerial fallout were consistently in the Cabrillo sampling location, located south-southeast of the Los Angeles area and Montrose property. These results were collected during non-Santa Ana wind conditions. Additional samples were collected during Santa Ana conditions. The Cabrillo sample location remained the highest in kg/month of aerial fallout; however, all areas

had a significant increase in the mass aerial fallout, with the Newport and San Clemente locations having the greatest relative increases.

Studies were conducted in the spring and fall of 1974 (Young et al., 1976a and b) to measure aerial fallout of DDT at 24 locations in the greater Los Angeles area (Figure 3-10). Samples were collected from the Montrose area (six sites), the areas surrounding each of two landfills that had previously accepted or still were accepting Montrose wastes (four sites each), and throughout the area (10 sites). Two regions were identified as having elevated DDT fallout: the area around the Montrose property and the area around the Rolling Hills Sanitary Landfill southwest of the Montrose property. The Rolling Hills Sanitary Landfill received DDT wastes from Montrose through 1972. Because the highest concentrations were detected southeast of each of these areas, and prevailing winds are from the west to northwest, these results were interpreted by Young to represent two separate sources. Seasonal effects were not seen in the data results.

#### **3.2.2.2 EPA-Lead Investigation**

Sampling of potential air releases near the Montrose property apparently was not conducted by Montrose during the years of operation. At the time of grading and capping, in 1985, Montrose conducted air monitoring; dust suppression techniques were also used during these activities. Montrose intended the air monitoring during capping activities to demonstrate that DDT was not being released to the air pathway (E&E, 1986).

In September of 1986, EPA contractor E&E conducted soil and dust sampling from areas surrounding the Montrose property to document historical air releases of DDT, DDE, and DDD from Montrose. Fifteen locations were sampled within a 1-mile radius. This study was intended to support the Hazard Ranking for inclusion of Montrose on the NPL. Samples were also collected by H + A as part of the off-

property soil study. Seventeen samples were collected in a semi-circular pattern around the property to the east, at approximately 0.5- and 0.75-mile radii (Figure 3-11).

Based on prevailing winds from the west and west-southwest 45 percent of the time for the period 1965 to 1974, sampling was conducted primarily to the north, east, and southeast of the property, but samples were also taken to the south and southwest. Background samples were collected at several locations, several miles west of Montrose.

E&E's concentration contours for p,p'-DDT in soil (Figure 3-12) were not clearly evident, and several general patterns could be observed in the soil sample results:

- The highest concentrations of DDT in soil samples were detected within several blocks of the Montrose property, to the east and northeast. Directly east of the property, on Jon Street, DDT was detected in soil at 98 mg/kg. To the northeast, along Francisco Street, DDT soil concentrations ranged from 18 to 37 mg/kg.
- Southeast and southwest of Montrose, there is little consistency in DDT concentrations for samples taken within several hundred feet of each other. To the southeast, along 204th Street, concentrations range from 1.8 to 21 mg/kg of DDT. To the southwest, along Del Amo Boulevard and 204th Street, DDT concentrations in soil range from 3.2 to 30 mg/kg.
- Samples collected from the south side of Torrance Boulevard, to the southeast and south, were all below 3 mg/kg. Half of these were within the range of DDT concentrations detected in background samples (1.5 mg/kg or lower). A single sample collected north-northeast of the property, along Knox Street, contained 2.2 mg/kg of DDT.

Dust sampling results for 4,4'-DDT generally exhibited a pattern similar to the soil results (Figure 3-13). The highest concentration was from a sample taken on Jon Street, located directly east of the property (250 mg/kg). Concentrations in samples from northeast of the property were inconsistent, ranging from 12 to 44 mg/kg. All samples exceeded the background concentrations by five to 150 times (E&E, 1986).

### 3.3 Identification of Chemicals of Potential Concern

In selecting those chemicals to be assessed in this ecological risk assessment, consideration was given to chemicals expected from Montrose activities and chemicals detected in the Montrose study area. As described in previous sections, the DDT formulation process used at Montrose involved chlorobenzene, chloral, sulfuric acid, caustic, and other compounds (Table 3-9). Impurities associated with the raw materials could have included di-, tri-, and hexachlorobenzenes (associated with chlorobenzene), and PCE, TCE, DCE, and DCA associated with chloral (also known as trichloroethanol). The product, technical grade DDT, contained both the "ortho, para-" and "para, para-" isomers (o,p' and p,p'-) of DDT, DDE, and DDD. Lubricants and solvents associated with the maintenance and operations of the facility would also be expected; however, no documentation of these was found. Chemicals detected on-property and in samples from surrounding areas were discussed. Table 3-1 summarizes this information.

Using available information, 17 chemicals were selected to be carried through the ecological risk assessment. Table 3-9 presents these chemicals, along with information on their association with the operations and their detected presence during previous investigations. Three frequently detected chemicals were not retained because of the potential for them to be laboratory contaminants; these are acetone, 2-butanone (i.e., methyl ethyl ketone) and methylene chloride. They also were not generally reported in environmental media where ecological receptors could be exposed to them, although acetone may have been present in groundwater or deeper soil samples. Other chemicals

**Table 3-9**  
**Organic Chemicals Detected in Soil from**  
**Various Investigations of the Montrose Chemical Plant**

Chemical	Central Process Area	Other On-Property Areas	Off-Property Areas	Raw Material	Products and By-products	Retained for Further Analysis
DDT	X	X	X		X	Yes
DDE	X	X	X		X	Yes
DDD	X	X	X		X	Yes
alpha-BHC	X	X	X		X	Yes
beta-BHC	X	X	X		X	Yes
delta-BHC	X	X	X		X	Yes
gamma-BHC	X	X	X		X	Yes
Acetone	X	X	X	a		-- <sup>c</sup>
Benzene	X	X		b		Yes
Benzoic Acid	X			a		--
Carbon tetrachloride	X					--
Chloral				X		Yes
Chlorobenzene	X	X	X	X		Yes
p-CBSA					X	Yes
Chloroform	X	X	X		X	Yes
Chlorophenols	X					--
Dichlorobenzene isomers	X	X				Yes
1,2-DCA	X			b		Yes
1,2-DCE	X			a		--
Ethylbenzene	X			a		Yes
Hexachlorobenzene	X			a		--
Hexachloroethane	X			b		--
Methylene chloride	X	X	X	a		c
2-Butanone	X	X		a		c
Phthalates	X	X	X	X		--
PCE	X	X	X	X		--
PNAs			X			--
Sodium hydroxide						--
Styrene	X			X		--
Sulfuric acid				X		--
Toluene	X	X	X	a		Yes
1,2,4-Trichlorobenzene	X			b		--
TCE			X	a		--
Xylenes	X	X	X	a		Yes

<sup>a</sup>Potential site maintenance solvents

<sup>b</sup>Potential impurities in chlorobenzene

<sup>c</sup>Potential laboratory contaminants

were not retained either because their association with Montrose activities is not clear (e.g., PCE, TCE) or there are inadequate data to determine if they are present on- or off-property (e.g., chlorophenols or hexachlorobenzene).

DRAFT

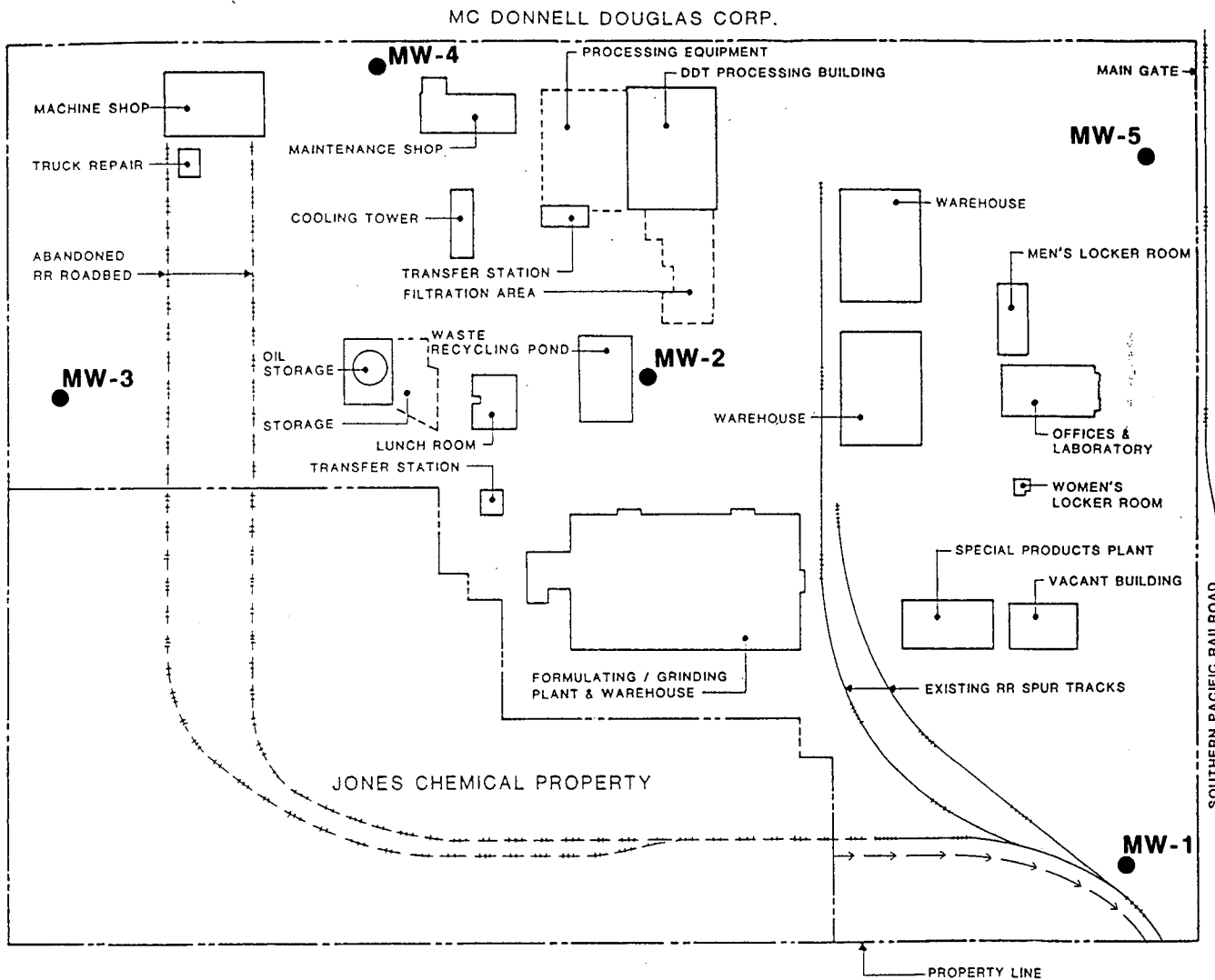
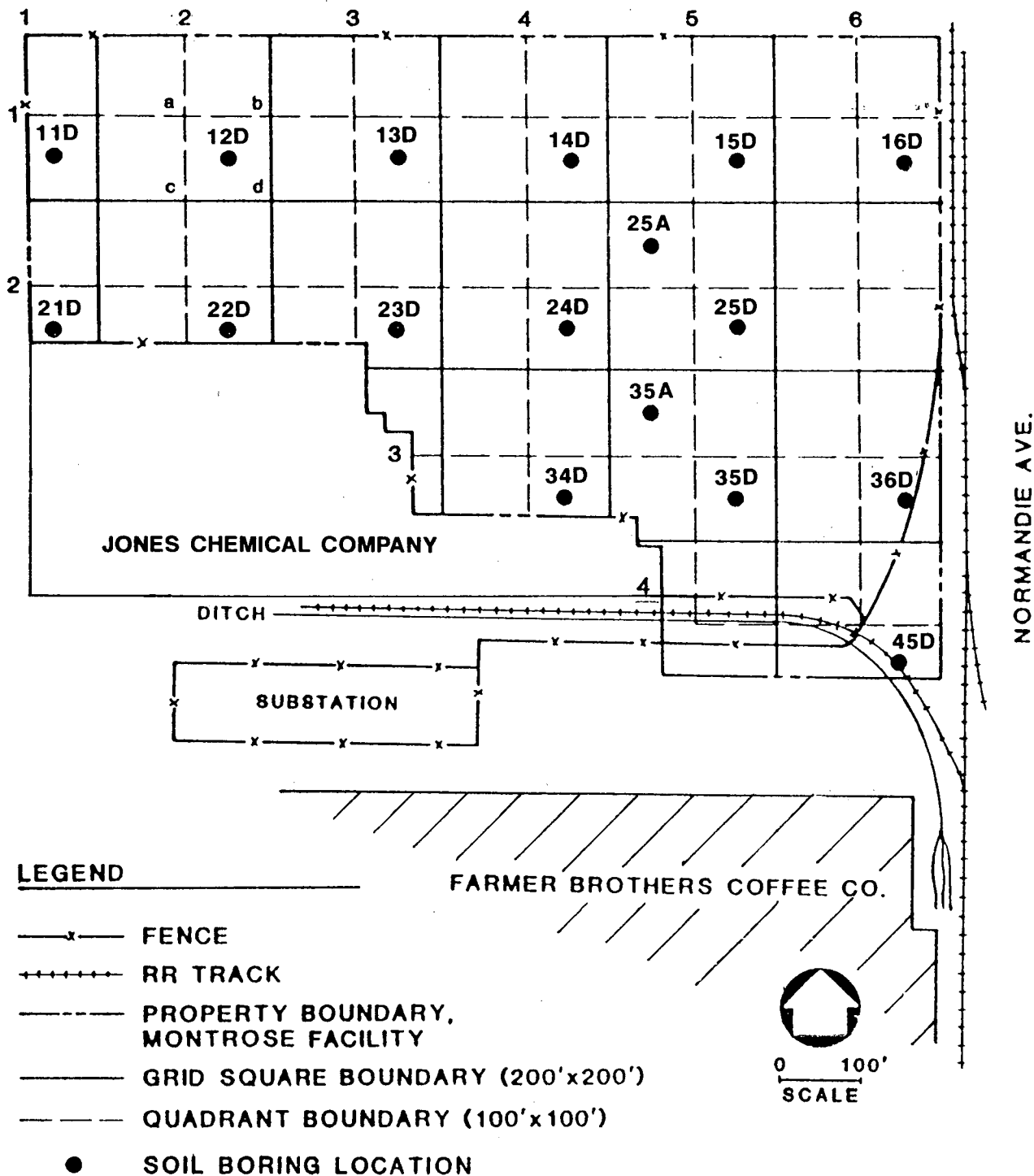


FIGURE 3-1  
LOCATION OF  
MONTROSE GROUNDWATER WELLS  
AND HISTORIC FACILITIES  
Ecological Risk Assessment  
Montrose Superfund Site

Source: Metcalf & Eddy, 1986



# McDONNELL DOUGLAS CORPORATION



Source: Metcalf & Eddy, 1986

FIGURE 3-2  
SOIL BORING LOCATIONS  
U.S. EPA RI INVESTIGATION, 1985  
MONTROSE CHEMICAL COMPANY  
Ecological Risk Assessment  
Montrose Superfund Site



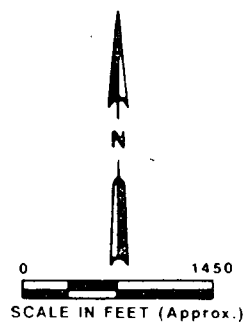
# LEGEND

KD-4.

Sample Name

FIGURE 3-3  
SEDIMENT IN  
THE KENWOOD DRAIN

Ecological Risk Assessment  
Montrose Superfund Site



SOURCE: Hargis + Associates, 1990

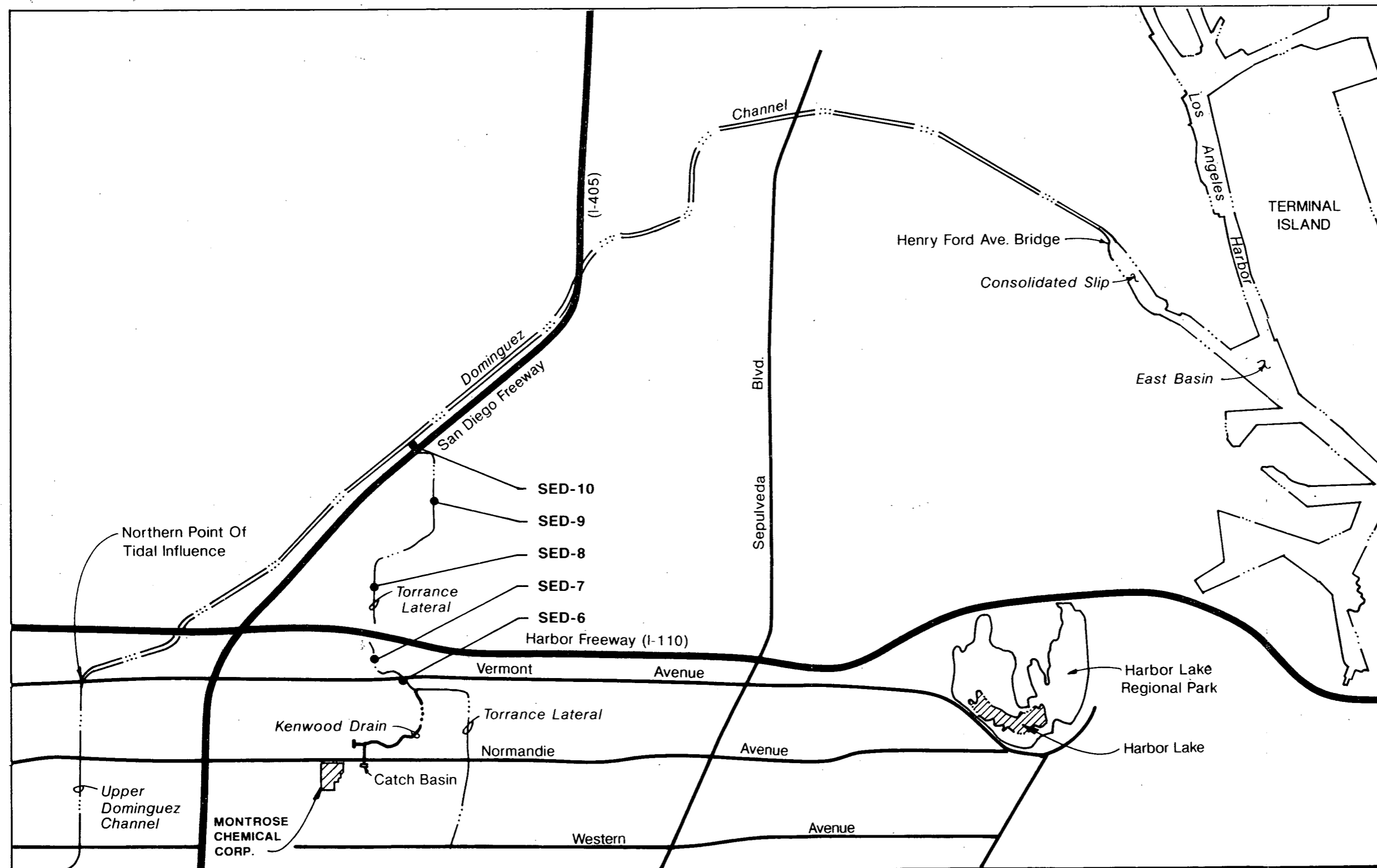
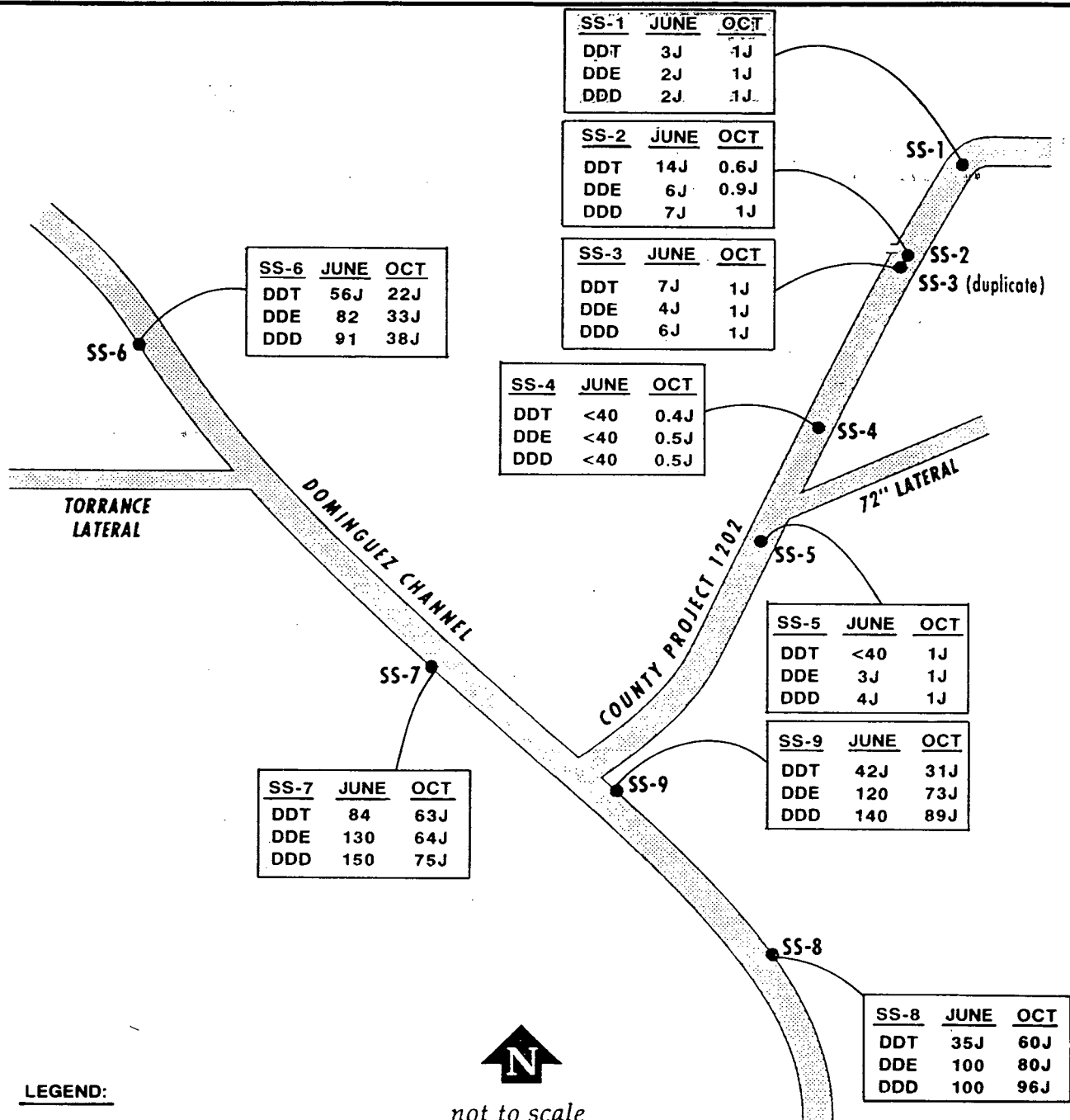


FIGURE 3-4  
SEDIMENTS IN TORRANCE LATERAL  
Ecological Risk Assessment  
Montrose Superfund Site

SOURCE: Hargis + Associates, 1990





SOURCE: Ecology & Environment, Inc. 1991

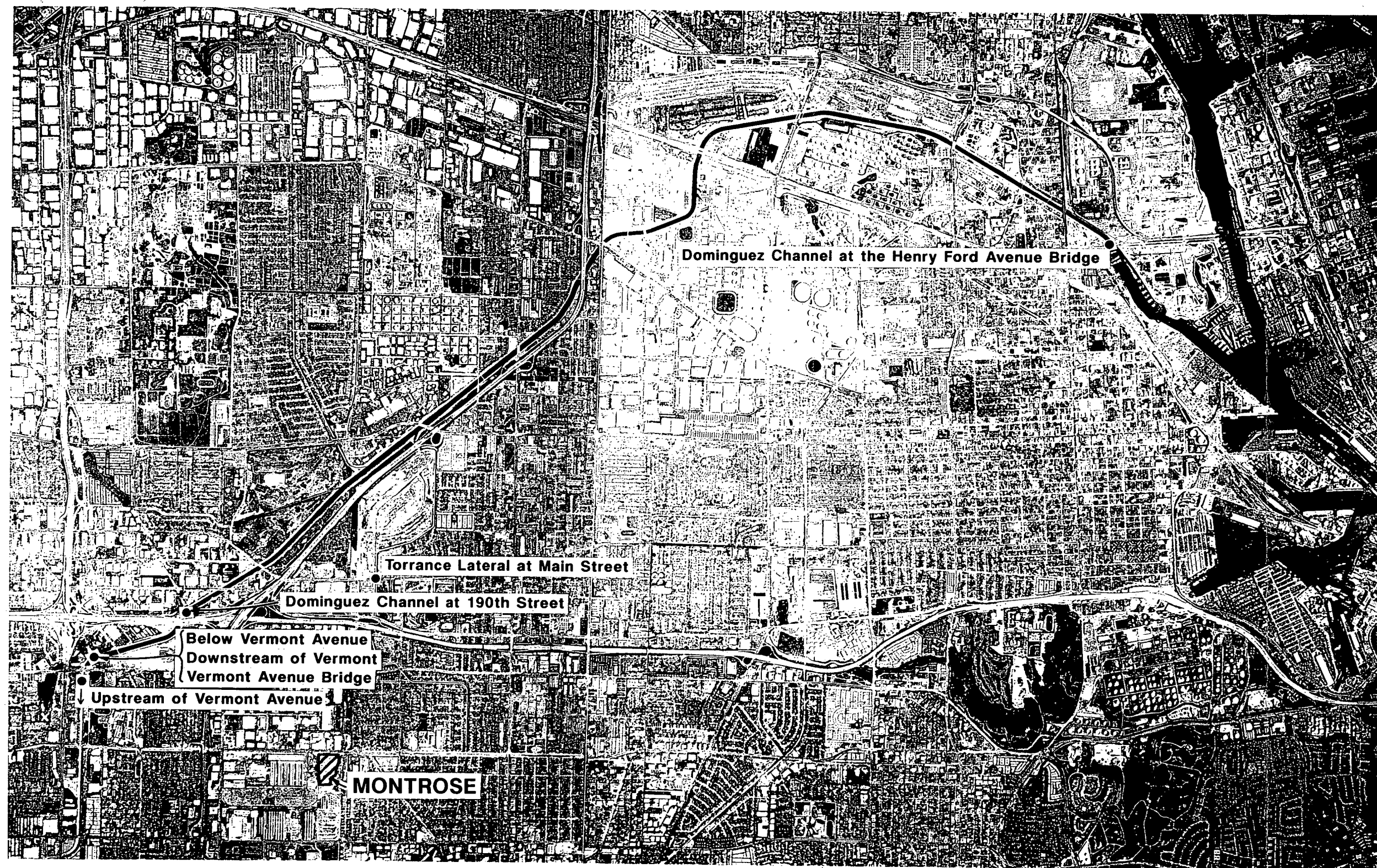


FIGURE 3-7  
 STORET DATA BASE  
 SAMPLE LOCATIONS IN THE  
 MONTROSE STUDY AREA  
 Ecological Risk Assessment  
 Montrose Superfund Site

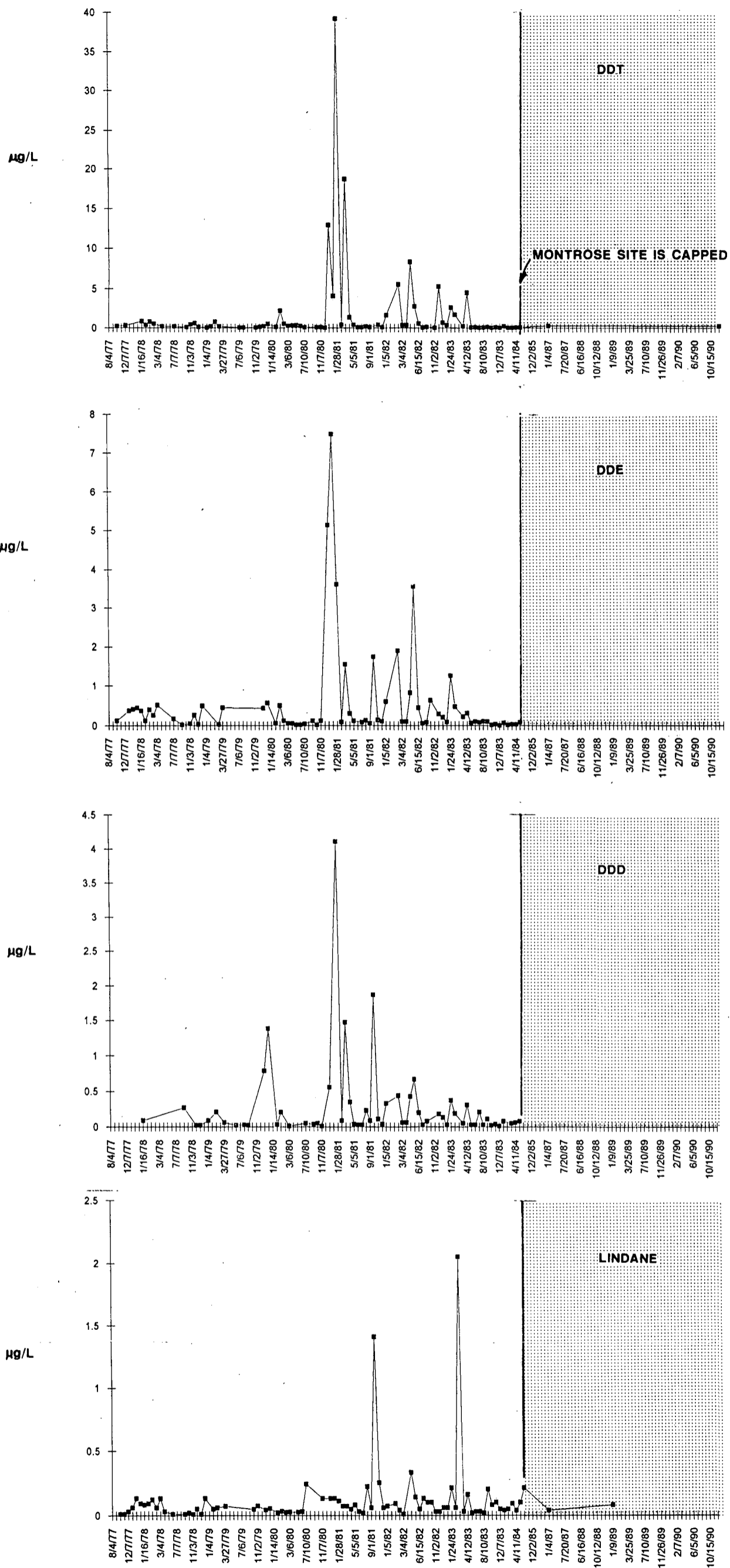
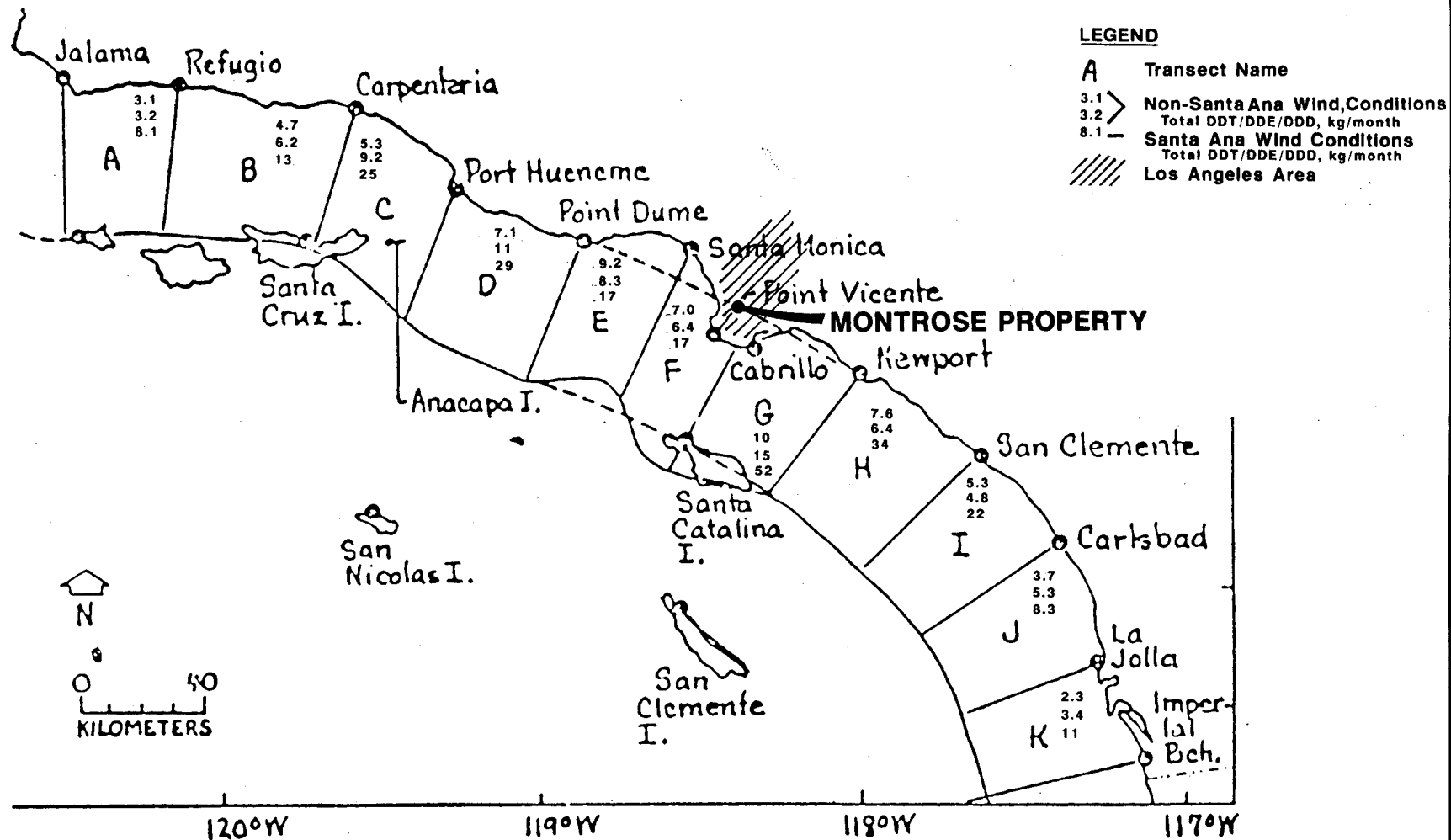
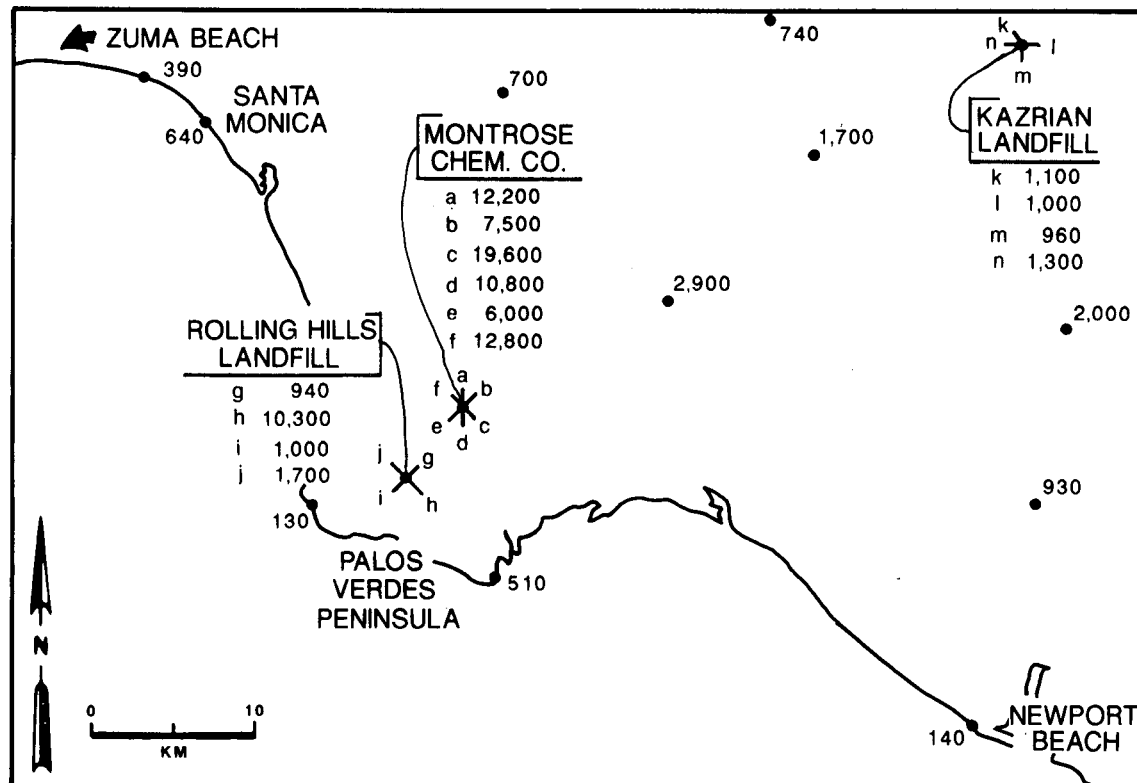


FIGURE 3-8  
STORET SURFACE WATER RESULTS FOR  
DDT, DDE, DDD AND LINDANE  
TORRANCE LATERAL AT MAIN STREET  
Ecological Risk Assessment  
Montrose Superfund Site



SOURCE: Young, et al., 1980.

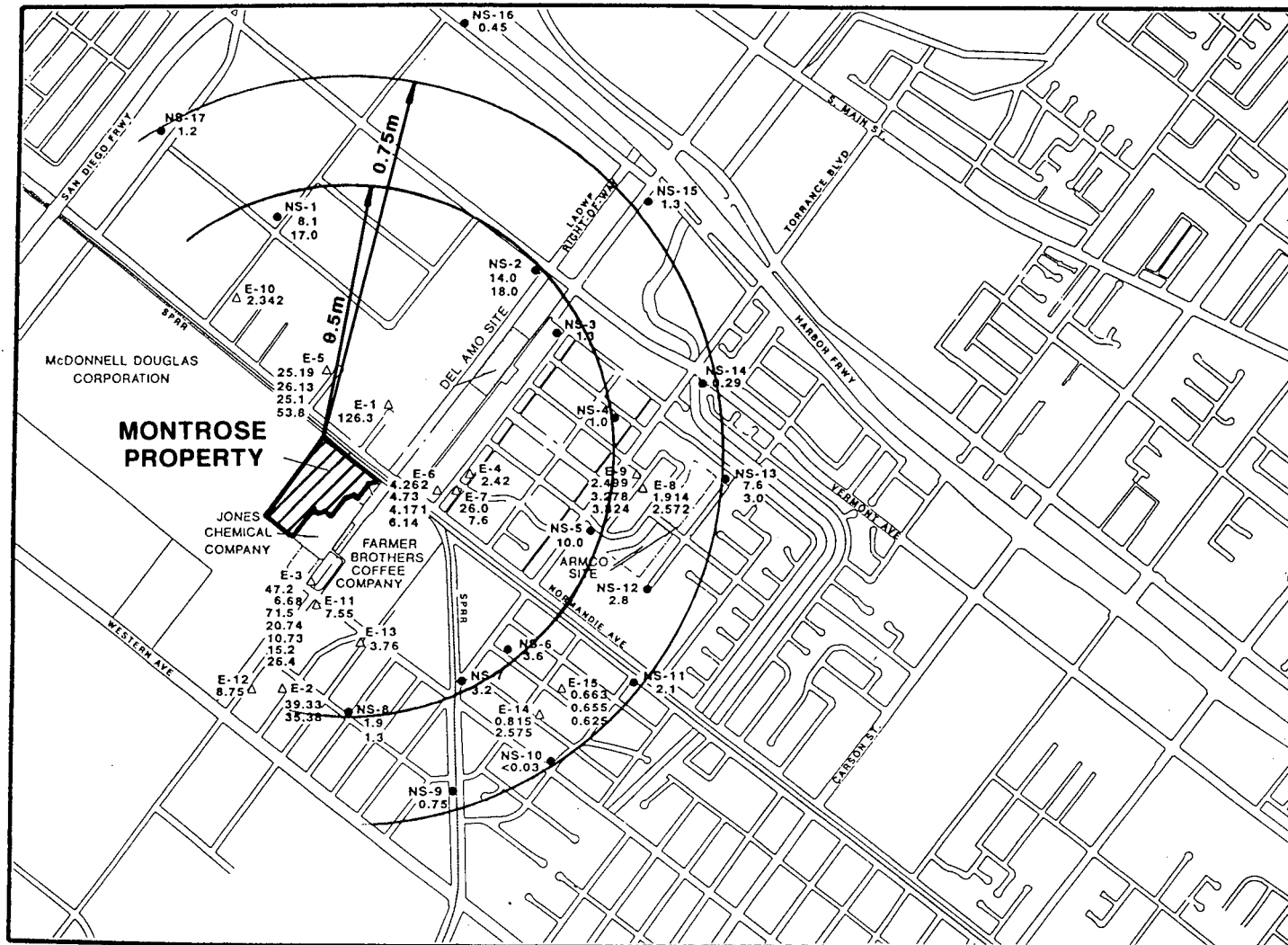
FIGURE 3-9  
MASS EMISSION RATE (kg/month) of  
DDT/DDE/DDD BY AERIAL FALLOUT  
Ecological Risk Assessment  
Montrose Superfund Site



SOURCE: Young et al., 1976 b

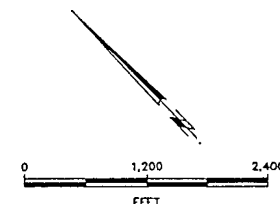
NOTE: Based on 6 weeks of sampling in 1974.

FIGURE 3-10  
 MEDIAN DRY AERIAL FLUX OF  
 TOTAL DDT ( $10^{-9}$  g/sq m/day)  
 Ecological Risk Assessment  
 Montrose Superfund Site



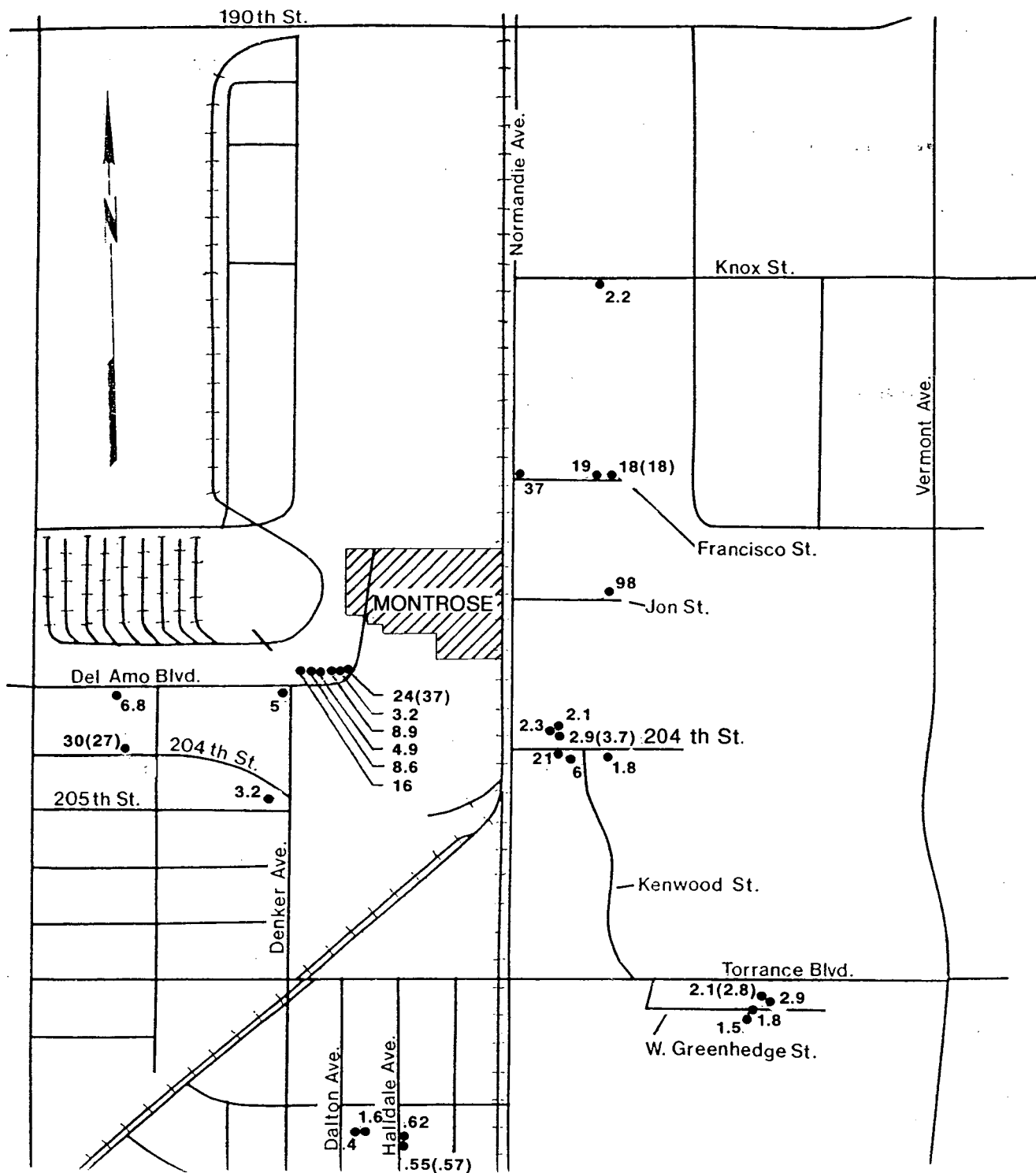
## EXPLANATION

- NS-14 MONTROSE SURFICIAL OFF-PROPERTY SOIL SAMPLE  
ORIGINAL AND FIELD DUPLICATE SAMPLE
- E-4 ECOLOGY AND ENVIRONMENT, INC. SURFICIAL OFF-PROPERTY  
SOIL SAMPLE, MULTIPLE SAMPLES COLLECTED AT EACH LOCATION
- NS-14  
0.29 TOTAL DDT CONCENTRATION  
IN MICROGRAMS PER KILOGRAM
- +++++ RAILROAD TRACKS
- SPRR SOUTHERN PACIFIC RAILROAD
- LADWP LOS ANGELES DEPARTMENT OF WATER AND POWER
- (<) = LESS THAN; NUMERICAL VALUE IS THE LIMIT  
OF DETECTION FOR THIS ANALYSIS



SOURCE: Hargis + Associates, 1990

**FIGURE 3-11**  
**DDT AERIAL DEPOSITION**  
**AROUND THE MONTROSE PROPERTY**  
Ecological Risk Assessment  
Montrose Superfund Site

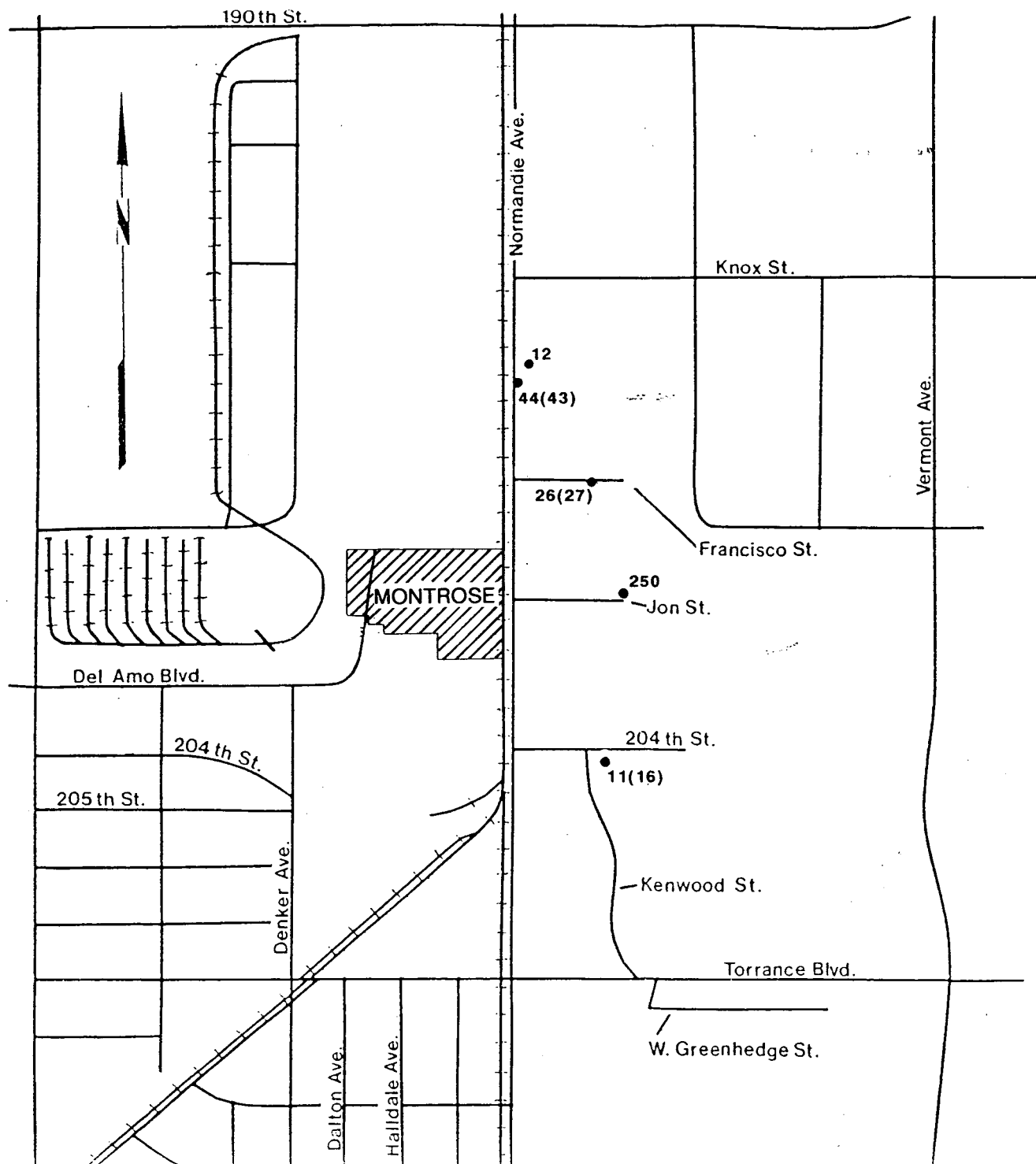


SOURCE: Ecology & Environment, 1986.

#### LEGEND

- Soil Sample Location
  - ( ) Duplicate
- Results in mg/kg

FIGURE 3-12  
E&E SOIL SAMPLE  
RESULTS FOR p,p'-DDT  
Ecological Risk Assessment  
Montrose Superfund Site



SOURCE: Ecology & Environment, 1986.

**LEGEND**

- Dust Sample Location
- ( ) Duplicate
- Results in mg/kg

FIGURE 3-13  
E&E DUST SAMPLE  
RESULTS FOR p,p'-DDT  
Ecological Risk Assessment  
Montrose Superfund Site

DRAFT

## 4 Ecological Receptors

## Section 4

### Ecological Receptors

#### 4.1 Aquatic Habitats

The aquatic habitats potentially affected by Montrose surface drainage include the Jones Ditch, Normandie Ditch, Kenwood Drain, Torrance Lateral, Dominguez Channel, and Consolidated Slip. Only the lower portion of the Torrance Lateral, the Dominguez Channel, and Consolidated Slip remain wet during dry-weather periods (dry weather is the dominant condition in this area). Most of the Torrance Lateral is an intermittent drainage, and the dry-weather wetted portion is very limited (H+A, 1990). The Torrance Lateral does not provide significant freshwater habitat for aquatic or semi-aquatic species. Therefore, the emphasis of this analysis of exposure and risk to aquatic organisms is on the Dominguez Channel and Consolidated Slip, both of which provide estuarine or marine habitat.

Organic contaminants transported from the Montrose property in runoff, including the upstream drainage channels that are usually dry, have been carried and stored within the channels, primarily in particle- and sediment-associated form. Sediments overlying concrete in the Torrance Lateral are typically nonexistent to a few inches thick, while sediments in the Dominguez Channel have accumulated up to 5.6 feet thick over a clay lining (H+A, 1990). There is a significant accumulation of sediment from the Torrance Lateral drainage at the junction with the Dominguez Channel (up to 2 feet thick). The Consolidated Slip sediments are soft, organic-rich sediments composed of dominant fractions of silt and clay (H+A, 1990; Soule and Oguri, 1980). An average of two samples taken at the mouth of the Dominguez Channel in the Consolidated Slip during 1978 indicated sediments of 18 percent sand, 55 percent silt, and 27 percent clay (Soule and Oguri, 1980). This sediment averaged 1.67 percent TOC and contained

nondetectable concentrations of total DDT. Although the detection limits are uncertain, concentrations as low as 0.0005 mg/kg total DDT were reported in the study.

The Dominguez Channel and Consolidated Slip are generally representative of the upper Los Angeles Harbor system with tidal influence and little water exchange to the open bay and ocean. The system is basically marine, with salinities that decrease with distance upstream along the Dominguez Channel. Although infrequent stormwater flows may create estuarine conditions in the upper Channel, marine species dominate the community of the Channel and Slip (as described in the following sections). The Dominguez Channel is concrete-lined in its upper portions, but in the reach downstream of Vermont Avenue, it is riprap lined on the edges with a clay bottom. The width at water surface is approximately 50 feet, and water depths are from 7 to 12 feet. The Consolidated Slip is wider and deeper than the Dominguez Channel (300 feet wide, 10 to 33 feet deep), with a soft mud bottom and concrete lining or rip-rap along most of the bank (H+A, 1990).

Both the Dominguez Channel and Consolidated Slip contain open water plankton and fish habitat, rocky or concrete intertidal zones, muddy benthic substrate, and open, deep water suitable for swimming and diving birds. Shallow, muddy areas that are used by shorebirds for wading and feeding have formed at both ends of the Consolidated Slip and at the entrance of the Torrance Lateral to the Dominguez Channel. The intertidal zone along the Dominguez Channel (and especially near the Torrance Lateral) also was used as a feeding area by shorebirds and wading birds during the reconnaissance surveys. There is little or no vegetation in the Dominguez Channel or Consolidated Slip.

## **4.2 Aquatic and Semi-aquatic Species**

The aquatic species of concern are those found in habitats that receive drainage from the Montrose property. Areas for investigation include the entire course of the Montrose drainage, except the upper portions near the property (portions of the Kenwood Drain), which are underground and hence not considered areas of immediate biological exposure (although they may be a reservoir for contaminants in sediments). Only the areas with potentially significant contaminant concentrations in sediment and dry-weather flows are included as aquatic habitats for species of concern. These areas are the marine and estuarine habitats of the Dominguez Channel and Consolidated Slip. The communities potentially exposed directly or indirectly (i.e., through the food chain) to contaminant releases from the Montrose property are composed of planktonic and attached algae, free-swimming and benthic invertebrates, fish, and birds.

### **4.2.1 Torrance Lateral and Upstream Drainage Channels**

The main Torrance Lateral drainage is an intermittent freshwater channel. It is a concrete-lined drainage with a flat bottom that is dry most of the time (H+A, 1990). However, the area of the Torrance Lateral immediately upstream from the Dominguez Channel contains saline water backed up from the Dominguez Channel. In general, the Montrose drainage system upstream from the Dominguez Channel (Torrance Lateral, Kenwood Drain) should not constitute a risk of exposure to aquatic organisms because the drainage channels are usually dry and therefore do not support aquatic communities.

### **4.2.2 Dominguez Channel**

The Dominguez Channel is tidally influenced and varies in salinity in relation to its distance upstream from Los Angeles Harbor and whether recent stormwater flows have entered into the upper channel. The biological community was characterized in 1972

and 1975 as basically marine/estuarine (Table 4-1). The benthic invertebrate community was dominated by capitellid polychaetes, which are considered indicators of polluted environments. Topsmelt and anchovy, the dominant fish (Table 4-1), are generally typical of the Southern California bay environment (Miller and Lea, 1972).

During the reconnaissance survey of the Channel on May 8, 1992, salinities ranged from near that of seawater (32.5 parts per thousand [ppt]) at the Consolidated Slip to 24.5 ppt at the Main Street overcrossing, upstream of the junction with the Torrance Lateral. Brackish water species normally occur at lower salinities than were observed in the Channel (Goldman and Horne, 1983). Marine benthic invertebrates (barnacles, tubed polychaetes) were observed attached to the rock lining throughout the Channel, indicating that average salinity conditions in the Dominguez Channel are marine. Filamentous algae, which can indicate high nutrient loading, were observed at a few locations, although the open water and channel edges were not generally enriched in algal growth.

Semi-aquatic birds were found throughout the Dominguez Channel during the field surveys (Table 4-2). Because of apparent differences in bird distribution and potentially different exposure levels in different portions of the Dominguez Channel, observations were recorded for three segments of the channel (Figure 4-1):

- Segment A included the portion from Broadway (about 1.5 miles northwest [upstream] from the Torrance Lateral) to Carson Street (about 1 mile southeast [downstream] from the Torrance Lateral).
- Segment B included the portion from Carson Street to Sepulveda Boulevard (about 2.5 miles).
- Segment C included the portion from Sepulveda Boulevard to the Consolidated Slip (about 2 miles).

**Table 4-1**  
**Biological Resources in the Dominguez Channel**  
**Near the ARCO Watson Refinery**

Species	Abundance <sup>a</sup>
<b>Fishes<sup>b</sup></b>	
<i>Anchoa compressa</i> (deepbody anchovy)	62
<i>Atherinops affinis</i> (topsmelt)	1,649
<i>Clevelandia ios</i> (bay goby)	6
<i>Cymatogaster aggregata</i> (shiner surfperch)	198
<i>Engraulis mordax</i> (northern anchovy)	7
<i>Fundulus parvipinnis</i> (California killifish)	L
<i>Gambusia</i> sp. (mosquitofish)	P
<i>Phanerodon furcatus</i> (white surfperch)	L
<i>Seriphus politus</i> (queenfish)	2
<b>Invertebrates<sup>c</sup></b>	
<u>Benthic</u>	
<i>Capitella capitata</i> (polychaete)	83
<i>Polydora lignia</i> (polychaete)	78
<i>Pseudopolydora paucibranchiata</i> (polychaete)	55
<i>Boccardia proboscidea</i> (polychaete)	1
<i>B. sp.</i> (polychaete)	1
<i>Nerinides?</i> sp. (polychaete)	5
<i>Cirriformia spirabrancha</i> (polychaete)	39
<i>Chaetozane</i> sp. (polychaete)	5
Oligochaeta	P
<i>Mytilus edulis</i> (bay mussel)	5
<i>Corophium acherusicum</i> (amphipod)	34
<i>C. insidiosum</i> (polychaete)	1
<i>Caprella</i> sp. (polychaete)	1
<i>Mesolamprops bispinosa</i>	3
<i>Ascidia ceratodes</i> (tunicate)	2
<u>Intertidal</u>	
<i>Balanus</i> spp. (barnacle)	C
<i>Eupomatis gracilis</i> (polychaete)	F
Unidentified polychaete tubes ( <i>Polydora?</i> ) (polychaete)	C
<i>Haminoea vesicula</i> (bubble shells)	C
<i>Hemigrapsus oregonensis</i> (mud crab)	2
Unidentified amphipods ( <i>Corophium</i> sp.?) (amphipod)	C
<i>Mytilus edulis</i> (bay mussel)	C/F
<i>Protothaca staminea</i> (littleneck clam)	1

<sup>a</sup>P = present; L = likely; C = common, numerous; F = few, scattered individuals or colonies.

<sup>b</sup>Adapted from Truesdale Laboratories, Inc. (1972); fishes seined during 12 monthly collections.

<sup>c</sup>Adapted from Jarvis (1975); intertidal and benthic invertebrates observed or collected in July 1975.

Source: CEC, 1986

**Table 4-2**  
**Semi-aquatic and Terrestrial Wildlife Species Expected to Occur in the Montrose Study Area**  
**and Those Observed During Field Surveys in February and May 1992**

Expected Species <sup>a</sup>	Observed—February 1992					Observed—May 1992				
	Dominguez Channel			Consolidated Slip	Terrestrial	Dominguez Channel			Consolidated Slip	Terrestrial
	A	B	C			A	B	C		
Amphibians										
Southwestern Toad										
Western Toad										
Pacific Treefrog										X
California Treefrog										
Bullfrog										
Reptiles										
Western Fence Lizard										
Gopher Snake										
Common Kingsnake										
Western Aquatic Garter Snake										
Lyre Snake										
Western Rattlesnake										
Birds										
Common Loon <sup>b</sup>										
Pied-billed Grebe	X	X	X	X		X				
Eared Grebe			X	X		X				
Western Grebe	X	X		X		X	X		X	
Clark's Grebe										
Brown Pelican <sup>b</sup>										
Double-crested Cormorant <sup>b</sup>	X	X	X	X		X		X		
Brandt's Cormorant										
Pelagic Cormorant										
American Bittern										
Least Bittern <sup>b</sup>										
Great Blue Heron			X							
Great Egret										
Snowy Egret	X									
Cattle Egret										
Green-backed Heron		X		X		X			X	
Black-crowned Night Heron								X		
Greater White-fronted Goose										
Snow Goose										
Brant										
Canada Goose										
Wood Duck										
Green-winged Teal										
Blue-winged Teal										

Continued

Continued

**Table 4-2**  
**Semi-aquatic and Terrestrial Wildlife Species Expected to Occur in the Montrose Study Area**  
**and Those Observed During Field Surveys in February and May 1992**

Expected Species <sup>a</sup>	Observed—February 1992					Observed—May 1992				
	Dominguez Channel			Consolidated Slip	Terrestrial	Dominguez Channel			Consolidated Slip	Terrestrial
	A	B	C			A	B	C		
Cinnamon Teal										
Mallard	X	X				X		X		
Northern Pintail										
Gadwall										
Eurasian Wigeon										
American Wigeon										
Canvasback										
Redhead										
Greater Scaup										
Lesser Scaup		X								
Oldsquaw										
Black Scoter										
Surf Scoter										
White-winged Scoter										
Common Goldeneye										
Bufflehead										
Hooded Merganser										
Red-breasted Merganser			X	X		X				
Common Merganser										
Ruddy Duck	X									
Turkey Vulture	X									
Osprey <sup>b</sup>										
Black-shouldered Kite										
Northern Harrier <sup>b</sup>										
Sharp-shinned Hawk <sup>b</sup>										
Cooper's Hawk <sup>b</sup>										
Red-shouldered Hawk										
Red-tailed Hawk										X
Ferruginous Hawk										
Bald Eagle <sup>b</sup>										
American Kestrel										
Merlin <sup>b</sup>										
Peregrine Falcon <sup>b</sup>										
Prairie Falcon										
California Quail										
Clapper Rail <sup>b</sup>										
Common Moorhen										
American Coot	X									

Continued

**Table 4-2**  
**Semi-aquatic and Terrestrial Wildlife Species Expected to Occur in the Montrose Study Area**  
**and Those Observed During Field Surveys in February and May 1992**

Expected Species <sup>a</sup>	Observed—February 1992					Observed—May 1992				
	Dominguez Channel			Consolidated Slip	Terrestrial	Dominguez Channel			Consolidated Slip	Terrestrial
	A	B	C			A	B	C		
Black-bellied Plover	X									
Snowy Plover <sup>b</sup>										
Semi-palmated Plover				X						
Killdeer	X									
Black-necked Stilt				X						
American Avocet				X						
Greater Yellowlegs	X									
Willet	X		X	X						
Wandering Tattler										
Spotted Sandpiper	X			X			X	X		
Whimbrel										
Long-billed Curlew										
Marbled Godwit										
Ruddy Turnstone										
Black Turnstone										
Sanderling										
Western Sandpiper	X			X						
Least Sandpiper	X			X						
Dunlin										
Long-billed Dowitcher	X									
Common Snipe										
Bonaparte's Gull										
Heermann's Gull						X				
Mew Gull										
Ring-billed Gull				X						
California Gull										
Herring Gull				X						
Thayer's Gull										
Western Gull			X	X					X	
Glaucous-winged Gull										
Caspian Tern										
Royal Tern										
Elegant Tern <sup>b</sup>										
Common Tern										
Forster's Tern										
Least Tern <sup>b</sup>										
Common Murre										
Rhinoceros Auklet <sup>b</sup>										

Continued

**Table 4-2**  
**Semi-aquatic and Terrestrial Wildlife Species Expected to Occur in the Montrose Study Area**  
**and Those Observed During Field Surveys in February and May 1992**

Expected Species <sup>a</sup>	Observed—February 1992					Observed—May 1992				
	Dominguez Channel			Consolidated Slip	Terrestrial	Dominguez Channel			Consolidated Slip	Terrestrial
	A	B	C			A	B	C		
Rock Dove <sup>b</sup>	X		X	X		X	X	X	X	X
Band-tailed Pigeon										
Spotted Dove	X									X
Mourning Dove	X	X			X	X	X	X		X
Common Barn Owl										
Burrowing Owl										
Short-eared Owl <sup>b</sup>										
White-throated Swift										
Black-chinned Hummingbird										
Anna's Hummingbird										X
Costa's Hummingbird										
Allen's Hummingbird										
Belted Kingfisher										
Red-breasted Sapsucker										
Northern Flicker										
Pacific-slope Flycatcher										
Black Phoebe										X
Say's Phoebe										
Horned Lark										
Cliff Swallow							X	X		
Barn Swallow	X						X	X		
Scrub Jay										
American Crow							X			X
Common Raven										X
Bushtit										X
Red-breasted Nuthatch										
White-breasted Nuthatch										
Rock Wren										
Canyon Wren										
Bewick's Wren										
House Wren										
Winter Wren										
Golden-crowned Kinglet										
Ruby-crowned Kinglet										
Western Bluebird										
Swainson's Thrush										
Hermit Thrush										
American Robin										

Continued

**Table 4-2**  
**Semi-aquatic and Terrestrial Wildlife Species Expected to Occur in the Montrose Study Area**  
**and Those Observed During Field Surveys in February and May 1992**

Expected Species <sup>a</sup>	Observed—February 1992					Observed—May 1992				
	Dominguez Channel			Consolidated Slip	Terrestrial	Dominguez Channel			Consolidated Slip	Terrestrial
	A	B	C			A	B	C		
Varied Thrush										
Wrentit										
Northern Mockingbird					X			X		X
California Thrasher										
American Pipit										
Cedar Waxwings										
Phainopepla										
Loggerhead Shrike	X					X		X	X	X
European Starling <sup>c</sup>	X					X	X	X	X	X
Orange-crowned Warbler										
Yellow Warbler <sup>b</sup>										
Yellow-rumped Warbler										
Townsend's Warbler										
Hermit Warbler										
Western Tanager										
Black-headed Grosbeak										
Lazuli Bunting										
Rufous-sided Towhee										
California Towhee										
Lark Sparrow										
Savannah Sparrow										
Fox Sparrow										
Song Sparrow										X
Golden-crowned Sparrow										
White-crowned Sparrow										
Dark-eyed Junco										
Red-winged Blackbird								X		
Tricolored Blackbird <sup>b</sup>										
Brewer's Blackbird										X
Brown-headed Cowbird										
Western Meadowlark	X									X
Hooded Oriole										
Northern Oriole										X
Purple Finch										
House Finch	X					X	X	X		X
Pine Siskin										
Lesser Goldfinch										
American Goldfinch										

Continued

**Table 4-2**  
**Semi-aquatic and Terrestrial Wildlife Species Expected to Occur in the Montrose Study Area**  
**and Those Observed During Field Surveys in February and May 1992**

Expected Species <sup>a</sup>	Observed—February 1992					Observed—May 1992				
	Dominguez Channel			Consolidated Slip	Terrestrial	Dominguez Channel			Consolidated Slip	Terrestrial
	A	B	C			A	B	C		
Lawrence's Goldfinch										
House Sparrow <sup>c</sup>	X					X				X
<b>Mammals</b>										
Yuma Myotis										
Long-eared Myotis										
Fringed Myotis										
Long-legged Myotis										
California Myotis										
Small-footed Myotis										
Western Pipistrelle										
Big Brown Bat										
Red Bat										
Hoary Bat										
Townsend's Big-eared Bat <sup>b</sup>										
Pallid Bat <sup>b</sup>										
Brazilian Free-tailed Bat										
Western Mastiff Bat <sup>b</sup>										
Desert Cottontail	X									
Brush Rabbit										
California Ground Squirrel						X	X	X		
Southwestern Pocket Gopher										X
Pacific Kangaroo Rat										
Western Harvest Mouse										
Deer Mouse										
California Vole										
Norway Rat										
Black Rat										
House Mouse										
Coyote										
Gray Fox										
Raccoon										
Long-tailed Weasel										
American Badger										
Striped Skunk										
California Sea Lion										
Harbor Seal										
Northern Elephant Seal <sup>b</sup>										

Continued

Table 4-2 Semi-aquatic and Terrestrial Wildlife Species Expected to Occur in the Montrose Study Area and Those Observed During Field Surveys in February and May 1992										
Expected Species <sup>a</sup>	Observed—February 1992						Observed—May 1992			
	Dominguez Channel			Consolidated Slip	Terrestrial	Dominguez Channel			Consolidated Slip	Terrestrial
	A	B	C			A	B	C		
<sup>a</sup> Source: CDFG, 1989. <sup>b</sup> Special-status species (i.e., listed as federal or state endangered or threatened, California species of special concern, or candidate for federal listing). <sup>c</sup> Species not protected by Migratory Bird Treaty Act; all other bird species are protected by that act.										

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Birds were observed throughout the Dominguez Channel, but the diversity and abundance were greatest in Segment A during the February surveys. In that area, grebes, cormorants, a snowy egret (*Egretta thula*), mallards (*Anas platyrhynchos*), mergansers, coots, and several species of shorebirds were observed feeding in proximity to the mouth of the Torrance Lateral, and most of the birds (a total of more than 100 individuals) were within about 100 yards of that site. During the May surveys, fewer than 10 birds were observed there each day.

#### 4.2.3 The Consolidated Slip

The Consolidated Slip is the best characterized of the potentially affected habitats in terms of water and sediment chemistry and biota. As one of the upper portions of Los Angeles Harbor, the Consolidated Slip has received some study as part of various recent harbor investigations (Soule and Oguri, 1980; Clark 1982). The area has changed significantly over the last 30 years as increasingly restrictive waste discharge limits have come into effect for the harbor and the Dominguez Channel. Pollution of the Consolidated Slip has decreased significantly from an era before 1962 when extended periods with no dissolved oxygen and toxic levels of hydrogen sulfide existed (Hertel, 1969). The pollution resulted from a number of discharges of organic and toxic wastes into the Dominguez Channel. The acute toxicity of the Dominguez Channel environment at that time eliminated many species that could be of concern for Montrose-related chronic contaminant exposure. The Consolidated Slip and other inner harbor communities were dominated by sulphur bacteria during the worst periods of anoxia and contamination (Soule and Oguri, 1980). The number of dischargers and degree of toxicity in the Dominguez Channel in the past make it impossible to separate Montrose drainage effects caused by toxicity from other discharges.

At this time (1992), the Consolidated Slip has recovered significantly and supports a marine flora and fauna characteristic of moderately polluted, mud bottom, tidal channels. Toxic contaminant concentrations are sufficiently low and dissolved oxygen

concentrations sufficiently high to support a varied community. The fish and benthic invertebrate communities of the harbor continue to evolve following the water quality improvements associated with the prevention of anoxia (Hertel, 1969) and reductions in system fertilization and productivity associated with recent decreases in wastewater discharge (Soule and Oguri, 1980).

Relatively recent species lists for fish and macro-invertebrates are given in Tables 4-3, 4-4, and 4-5. Because fish are highly mobile, Los Angeles Harbor surveys are representative of some of the Consolidated Slip community. No special-status species (fish or invertebrates) in the local aquatic community were noted in the 1982 (Table 4-3) and 1990 (Table 4-4) surveys. The fish and invertebrates were typical of the upper Los Angeles Harbor community (Soule and Oguri, 1980). The Consolidated Slip shows the effect of biostimulation of phytoplankton populations through the Dominguez Channel discharges; in comparison to other harbor locations, the Consolidated Slip often has relatively high algal biomass and productivity (Soule and Oguri, 1980). However, the lowest zooplankton diversity and abundance for Los Angeles Harbor were found in the inner harbor areas, including the Consolidated Slip.

The community of benthic organisms in the Consolidated Slip appeared to be more indicative of pollution impacts than those communities located toward the outer harbor, when examined in 1978. The predominance of capitellid polychaetes in the benthos was indicative of a moderately polluted, soft-sediment environment. The total number of benthic species collected in the Consolidated Slip ranged from 7 to 11 during 1978 quarterly samples with a mean density of 8,576 individuals per square meter (Soule and Oguri, 1980).

The double-crested cormorant (*Phalacrocorax auritus*) is a state species of special concern because of population declines throughout parts of its range in California (Remsen, 1978). This species was observed in the Consolidated Slip during February and in the Dominguez Channel during both surveys (February and May, 1992). The double-crested cormorant was the only special-status species observed in the study area

**Table 4-3**  
**Fish Species Found in Los Angeles-Long Beach Inner Harbors**  
**(1971-1979)**

Species Name <sup>a</sup>	Common Name
<i>Anchoa compressa</i>	Deepbody anchovy
<i>Anchoa delicatissima</i>	Slough anchovy
<i>Anisoremus davidsonii</i>	Sargo
<i>Artedius lateralis</i>	Smoothhead sculpin
<i>Atherinops affinis</i>	Topsmelt
<i>Atractoscion nobilis</i>	White seabass
<i>Cheilotrema saturnum</i>	Black croaker
<i>Chilara taylori</i>	Cusk eel
<i>Citharichthys sordidus</i>	Pacific sanddab
<i>Citharichthys stigmaeus</i>	Speckled sanddab
<i>Clevelandia ios</i>	Arrow goby
<i>Cymatogaster aggregata</i>	Shiner surfperch
<i>Cyprinus carpio</i>	Carp
<i>Damalichthys vacca</i>	Pile surfperch
<i>Embiotoca jacksoni</i>	Black surfperch
<i>Engraulis mordax</i>	Northern anchovy
<i>Genyonemus lineatus</i>	White croaker
<i>Gobiidae (unid.)</i>	Goby
<i>Heterostichus rostratus</i>	Giant kelpfish
<i>Hippoglossina stomata</i>	Bigmouth sole
<i>Hyperprosopon argenteum</i>	Walleye surfperch
<i>Ilypnus gilberti</i>	Cheekspot goby
<i>Lepidogobius lepidus</i>	Bay goby
<i>Leptocottus armatus</i>	Staghorn sculpin
<i>Leuresthes tenuis</i>	California grunion
<i>Mustelus henlei</i>	Brown smoothhound
<i>Myliobatis californica</i>	Bat ray
<i>Neoclinus uninotatus</i>	Onespot fringehead
<i>Odontopyxis trispinosa</i>	Pygmy poacher
<i>Otiophidium scrippsii</i>	Basketweave cusk-eel
<i>Paralabrax nebulifer</i>	Barred sand bass
<i>Paralichthys californicus</i>	California halibut

Continued

**Table 4-3**  
**Fish Species Found in Los Angeles-Long Beach Inner Harbors**  
**(1971-1979)**

Species Name <sup>a</sup>	Common Name
<i>Parophrys vetulus</i>	English sole
<i>Peprilus simillimus</i>	Pacific butterfish
<i>Phanerodon furcatus</i>	White surfperch
<i>Pleuronichthys decurrens</i>	Curlfin turbot
<i>Pleuronichthys riueri</i>	Spotted turbot
<i>Pleuronichthys verticalis</i>	Hornyhead turbot
<i>Porichthys myriaster</i>	Specklefin midshipman
<i>Porichthys notatus</i>	Plainfish midshipman
<i>Rhacochilus toxotes</i>	Rubberlip surfperch
<i>Rhinobatos productus</i>	Shovelnose guitarfish
<i>Scorpaena guttata</i>	Sculpin or spotted scorpionfish
<i>Sebastes auriculatus</i>	Brown rockfish
<i>Sebastes dalli</i>	Catco rockfish
<i>Sebastes miniatus</i>	Vermilion rockfish
<i>Sebastes mystinus</i>	Blue rockfish
<i>Sebastes paucispinis</i>	Bocaccio
<i>Sebastes saxicola</i>	Stripetail rockfish
<i>Sebastes semicinctus</i>	Halfbanded rockfish
<i>Sebastes serranoides</i>	Olive rockfish
<i>Sebastes sp. (unid.)</i>	Rockfish
<i>Seriphus polius</i>	Queenfish
<i>Squalus acanthias</i>	Spiny dogfish
<i>Symphurus atricauda</i>	California tonguefish
<i>Synodus lucioceph</i>	California lizardfish
<i>Syngnathus sp.</i>	Pipefish
<i>Trachurus symmetricus</i>	Jack mackerel
<i>Urophycis halleri</i>	Round stringray
<i>Xystreurys liolepis</i>	Fantail sole
<sup>a</sup> Names as presented in Soule and Oguri, 1980.	
Source: Soule and Oguri, 1980.	

**Table 4-4**  
**Benthic Community of Annelida (Segmented Worms)**  
**in the Vicinity of Berth 200Y in the Consolidated Slip**  
**Los Angeles Harbor**  
**(1982)**

*Boccardia proboscidea*  
*Capitella capitata*<sup>a</sup>  
*Eumida bifoliata*  
*Eumida sanguinea*  
Nereidae (juvenile)  
*Nereis procera*  
*Polydora socialis*  
*Psuedopolydora paucibranchiata*<sup>b</sup>  
*Pseudopolydora kempii californica*

Oligochaeta, unidentified

<sup>a</sup>Indicator organism for "polluted" area (Reish, 1959; Hill, 1974).

<sup>b</sup>Indicator organism for "semi-healthy" area (Reish, 1959).

Source: POLA, 1983.

**Table 4-5**  
**Dominant Fish Species**  
**Collected in Los Angeles-Long Beach Harbor**  
**(1990)**

Common Name	Scientific Name	Habitat	Gear
White croaker	<i>Genyonemus lineatus</i>	b,p	T,G,P,L
Queenfish	<i>Seriphus politus</i>	b,p	T,G,P,L,S
White surfperch	<i>Phanerodon furcatus</i>	b,p	T,G
Northern anchovy	<i>Engraulis mordax</i>	p	T,P,L,S
California tonguefish	<i>Symphurus atricauda</i>	b	T
California halibut	<i>Paralichthys californicus</i>	b	T
Speckled sanddab	<i>Citharichthys stigmaeus</i>	b	T
Shiner surfperch	<i>Cymatogaster aggregata</i>	b	T
Bay goby	<i>Lepidogobius lepidus</i>	b	T
Basketweave cusk-eel	<i>Ophidian scrippsae</i>	b	T
Black surfperch	<i>Embiotoca jacksoni</i>	b	G
Walleye surfperch	<i>Hyperprosopon argenteum</i>	b	G
Topsmelt	<i>Atherinops affinis</i>	p	S
Arrow goby	<i>Clevelandia ia</i>	b	S
Cheekspot goby	<i>Ilypnus gilberti</i>	b	S
Pacific sardine	<i>Sardinops sagax caeruleus</i>	p	P,L
Pacific butterfish	<i>Peprillus similimus</i>	p	P
California barracuda	<i>Sphyræna argentea</i>	p	P
Jack mackerel	<i>Trachurus symmetricus</i>	p	P
California grunion	<i>Leuresthes tenuis</i>	p	L,S
Jacksmelt	<i>Atherinopsis californiensis</i>	p	S

**Notes:**

- T = Abundant in otter trawl collections
- G = Abundant in gill net collections
- S = Abundant in beach seine collections
- P = Abundant in purse seine collection
- L = Lampera net collection
- b = Epibenthic-demersal
- p = pelagic

Source: Long Beach and Los Angeles Harbor Departments and the U.S. Army Corps of Engineers, Los Angeles District (1990).

during the reconnaissance surveys. Other special-status species that could be expected to occur in the study area are indicated in Table 4-2.

Other birds observed in the Consolidated Slip included grebes, herons, waterfowl, shorebirds, and gulls. These species (as well as cormorants) feed primarily on fish and benthic macro-invertebrates. The presence and observed feeding behavior of these birds indicate the availability of their food resources in this habitat. Semi-aquatic birds and mammals expected to occur and those observed during the field surveys are listed in Table 4-2.

General differences in current seasonal presence of the bird species are indicated by differences between observations in February and May 1992 (Table 4-2). Some birds were present only at the time of the winter surveys, whereas others were present at the time of the winter and spring surveys. In general, there were more wintering birds than breeding-season birds. Although the reconnaissance surveys were not designed to quantify populations present, some species (such as western grebes) were relatively abundant (30 or more individuals) during each day of the February survey.

### **4.3 Terrestrial Habitats**

The largest tracts of terrestrial habitats in the study area that could be important for wildlife species include less intensively developed areas east (downwind) from the Montrose property and north of the Torrance Lateral (some of which is currently being developed), as well as open habitats in the Dominguez Golf Course, Victoria Golf Course, and Goodyear Airship Field, and along the freeway rights-of-way in that vicinity (Figure 2-7). Vacant lots and residential yards also provide habitat for terrestrial wildlife.

These urbanized habitats include areas with mostly non-native vegetation under varying degrees of development and cultivation. They are found closer to the Montrose property than the aquatic habitats and could provide significant exposure points for terrestrial species if contaminants are found in the soil.

#### **4.4 Terrestrial Species**

Urbanized habitats are characterized by species that have adapted to human presence and disturbance. Terrestrial species associated with the study area habitats may use portions of the aquatic habitats as they forage in shallow water or they may forage over open water. These species include animals such as swallows foraging for flying insects over open water; mice living and foraging in and among rocky shorelines; and raccoons and skunks preying on a variety of insects, other animals, and plant material along the shoreline and in shallow water. Many species may forage and nest in the urbanized terrestrial habitat, including sparrows that forage for seeds, robins that forage for worms and other invertebrates, starlings and meadowlarks that eat many terrestrial insects, voles and mice that forage for vegetation and seeds, and hawks that prey on small vertebrates.

Terrestrial species of birds and mammals that were observed during the February and May 1992 field surveys are listed in Table 4-2. It should be noted that, because of the secretive or nocturnal behavior of many of the expected species and the limited extent of the reconnaissance surveys, the lack of observations for many species does not indicate their absence.

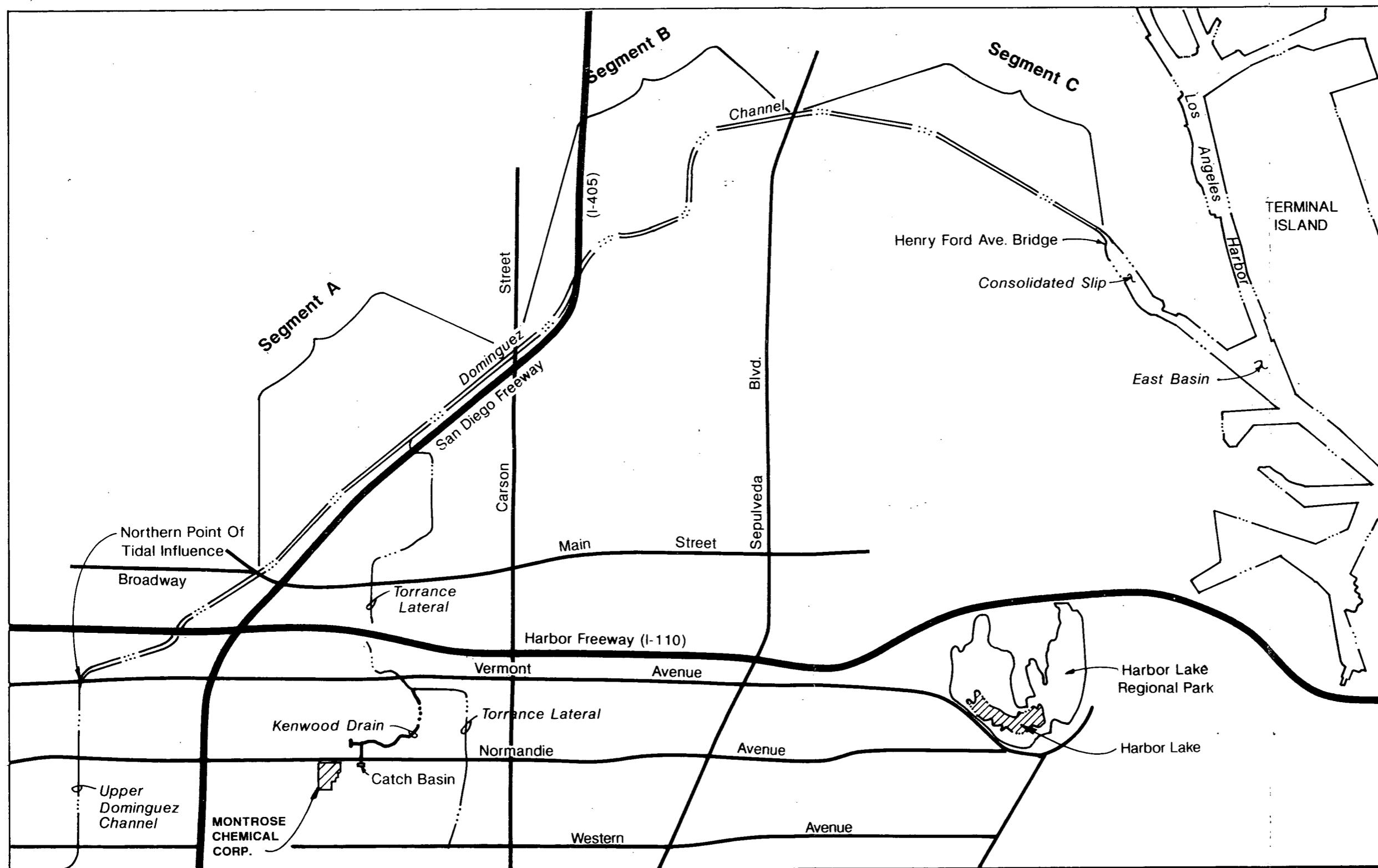


FIGURE 4-1  
SEGMENTS OF THE  
DOMINGUEZ CHANNEL  
SURVEYED FOR BIRDS  
Ecological Risk Assessment  
Montrose Superfund Site

## **5 Exposure Mechanisms and Pathways**

## Section 5

# Exposure Mechanisms and Pathways

### 5.1 Environmental Fate and Chemical Transport Mechanisms

Environmental contaminants may be transported from one medium to another by a variety of chemical, physical, and biological processes. The major processes involved in the chemical transport and conversion of contaminants in the environment include hydrolysis, volatilization, sorption, oxidation/reduction, biodegradation, and bioaccumulation. This section describes some of the processes that may occur in air, soil, water, and living organisms for the compounds identified in the study area. The chemical and physical characteristics of these contaminants that govern their mobility and biological fate are presented in Table 5-1. The table also includes a range of bioconcentration factors for aquatic species. Bioconcentration factors are presented in greater detail in Appendix A, which is based on data from the ASTER (1992) and HSDB (1992) databases.

#### 5.1.1 DDT, DDE, and DDD

Studies of DDT transformations in soils indicate prolonged persistence (Howard, 1991). DDT, DDE, and DDD have a high affinity for (tend to adsorb to) soil particles, as predicted by their soil adsorption partition coefficients (Table 5-1). These compounds are only slightly soluble in water. Mobility of DDT, DDE, and DDD during periods of runoff is primarily caused by transport of particulate matter to which these compounds are bound. Volatilization from near-surface soils and water is an important migration pathway for DDT and DDE. Their tendency to volatilize from water and soil surfaces can be predicted by their Henry's law constants and vapor pressures, respectively, and the organic content of the soil. The estimated half-life of volatilization for DDT is 100 days (Sleicher and Hopcraft, 1984). The tendency of DDD to volatilize is

**Table 5-1**  
**Characteristics of Selected Contaminants of Concern at the Montrose Study Area**

Chemical name	Mole Weight (g/mole)	Water Solubility (mg/l)	Vapor Pressure (mmHg)	Henry's Law Constant(II) $\frac{\text{atm}\cdot\text{m}^3}{\text{mole}}$	Octanol/Water Coefficient Log ( $K_{ow}$ )	Soil Adsorption Coefficient	BCF in Fish Tissue (l/kg)
DDT (total)	354.50 <sup>1</sup>	0.0031 - 0.0034 @ 25°C <sup>2</sup>	0.00000015 @ 20°C <sup>3</sup>	0.000028 @ 25°C <sup>4</sup>	6.36 <sup>5</sup>	320,000 <sup>6</sup>	38,000 (rainbow trout) <sup>7</sup> 110,000 (estimated) <sup>8</sup>
DDE (total)	318.02 <sup>1</sup>	0.04 @ 20°C <sup>2</sup>	0.0000066 - 6.2 at 20°C <sup>9</sup>	0.00019 at 25°C <sup>10</sup>	5.69 (p,p'-isomer), 5.78 (o,p'-isomer) <sup>9</sup>	257,000 <sup>11</sup>	110,000 (bluegill) <sup>22</sup>
DDD (total)	320.05 <sup>1</sup>	0.16 @ 24°C <sup>2</sup>	0.0000000013 - 0.0000000025 @ 30°C <sup>9</sup>	0.000031 @ 25°C <sup>10</sup>	5.56 <sup>6</sup>	240,000 <sup>6</sup>	174,000 (estimated) <sup>8</sup>
Lindane	290.85 <sup>1</sup>	7.80 @ 25°C <sup>9,12</sup>	0.00000094 @ 20°C <sup>13</sup>	0.00000048 @ 20°C <sup>14</sup>	3.72 <sup>9,15</sup>	2,500 (estimated) <sup>16</sup>	250 (estimated) <sup>8</sup>
Benzene	78.11 <sup>1</sup>	1,780 @ 20°C <sup>2</sup>	76 @ 20°C <sup>2</sup>	0.00543 @ 25°C <sup>17</sup>	2.13 <sup>15</sup>	65 <sup>8</sup>	6.5 (estimated) <sup>8</sup>
Chlorobenzene	112.56 <sup>1</sup>	490 @ 25°C <sup>18</sup>	8.8 @ 20°C <sup>19</sup>	0.00346 @ 25°C <sup>17</sup>	2.84 <sup>15</sup>	333 <sup>8</sup>	10.3 (est. for edible aquatic organisms) <sup>23</sup> 33 (estimated) <sup>8</sup>
Chloroform	119.39 <sup>1</sup>	8,220 at 20°C <sup>21</sup>	160 @ 20°C <sup>19</sup>	0.00375 @ 20°C <sup>17</sup>	1.97 <sup>15</sup>	4.40 <sup>12</sup>	6 (bluegill) <sup>24</sup> 4.5 (estimated) <sup>8</sup>
1,2-DCA	98.96 <sup>1</sup>	8,690 at 20°C <sup>21</sup>	63.7 @ 20°C <sup>21</sup>	0.0040 @ 25°C <sup>17</sup>	1.48 <sup>15</sup>	1.40 <sup>12</sup>	2 (bluegill) <sup>11</sup> 1.40 (estimated) <sup>25</sup>
Ethylbenzene	106.16 <sup>1</sup>	152 @ 20°C <sup>2</sup>	7.00 @ 20°C <sup>2</sup>	0.0079 @ 25°C <sup>17</sup>	3.15 <sup>15</sup>	681 <sup>8</sup>	6.80 (estimated) <sup>8</sup> 9.50 (estimated) <sup>26</sup>
Toluene	92.14 <sup>1</sup>	515 @ 20°C <sup>2</sup>	2.20 @ 20°C <sup>2</sup>	0.00661 @ 25°C <sup>17</sup>	2.73 <sup>15</sup>	2591 <sup>8</sup>	2.60 (estimated) <sup>8</sup> 2.71 (estimated) <sup>27</sup>
Xylene	106.17 <sup>1</sup>	0.30 @ 20°C <sup>19</sup>	9.00 @ 20°C <sup>19</sup>	0.00701 @ 25°C <sup>17</sup>	3.15 <sup>15</sup>	691 <sup>8</sup>	7.00 (ave. estimate for o-,m-, p-isomers) <sup>8</sup>

Continued

**Table 5-1**  
**Characteristics of Selected Contaminants of Concern at the Montrose Study Area**

Chemical name	Mole Weight (g/mole)	Water Solubility (mg/l)	Vapor Pressure (mmHg)	Henry's Law Constant(H) $\frac{\text{atm-m}^3}{\text{mole}}$	Octanol/Water Coefficient Log ( $K_{ow}$ )	Soil Adsorption Coefficient	BCF in Fish Tissue (l/kg)
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Notes:

1. BEIA, 1989.
2. Verschueren, K., 1983, as cited in (1).
3. Clayton, 1981, as cited in (1).
4. Lyman, 1985, and Neely et al., 1985, as cited in (1).
5. Chiou et al., 1982 as cited in (1).
6. Kadeg et al., 1986 as cited in (1).
7. EPA, 1980a, as cited in (1).
8. As cited in (1). Values were estimated by Authur D. Little, Inc. using  $K_{ow}$  as the basis of estimation.
9. Callahan et al., 1979, as cited in (1).
10. As cited in (1), value estimated using vapor pressor and solubility data from (9).
11. As cited in (1), values were estimated by Authur D Little, Inc., using the equation given by (12), which uses  $K_{ow}$  as a basis of estimation.
12. Maybe et al., 1981, as cited in (1).
13. Toxicology Data Bank (TDB) Database, 1984, as cited in (1).
14. Lyman and Reehl, 1982, as cited in (1).
15. Leo, 1983, as cited in (1).
16. Means et al., 1982, as cited in (1).
17. Mackay and Shiu, 1981, as cited in (1).
18. As cited in (1), values were estimated by Authur D Little, Inc.
19. Mackison et al., 1981, as cited in (1).
20. USEPA, 1980, as cited in (1).
21. Grayson, M.; Eckroth, D., eds, 1978.
22. EPA 1980b, as cited in (1).
23. EPA 1980c, as cited in (1).
24. EPA 1980d, as cited in (1).
25. EPA 1980e, as cited in (1).
26. EPA 1980f, as cited in (1).
27. EPA 1980g, as cited in (1).

approximately threefold less than that of DDT or DDE (ATSDR, 1989c). Laboratory studies of the air/water partition coefficient of DDE indicate that it will volatilize from seawater 10 to 20 times faster than from freshwater (Atlas et al., 1982).

Residues of volatilized or airborne DDT and its degradation products are removed from the atmosphere by precipitation, diffusion into large bodies of water, and chemical transformation. The photo-oxidation half-life of DDT, DDE, and DDD is about 7 days (Howard et al., 1991).

When released to water, DDT may be partitioned, transported or converted in several ways:

- Adsorption to sediments
- Bioconcentration in aquatic organisms
- Volatilization
- Photodegradation
- Biodegradation

DDT, DDE, and DDD are highly lipid-soluble and may be biomagnified in the food chain (ATSDR, 1989c).

Biodegradation is expected to be the predominant fate process in soils. In the presence of certain microorganisms, biodegradation is known to occur under anaerobic and aerobic conditions (HSDB, 1992). Under aerobic conditions, DDT is slowly converted to DDE, whereas under anaerobic conditions, DDT is converted more rapidly to DDD. Both DDD and DDE are very resistant to further degradation. The estimated half-life of DDT and its metabolites is 2 to >15 years. Hydrolysis is not expected to be significant. The photo-oxidation half-life of DDT in water is 7 to 350 days (Howard et al., 1991).

### 5.1.2 BHC and Lindane

Once released to the environment, BHC isomers, including lindane (gamma-BHC), can partition to all environmental media. In soil, lindane can leach to groundwater, sorb to soil particles, or volatilize to the atmosphere. The most important factor governing sorption is the organic matter content of the soils. Lindane may undergo hydrolysis and biodegradation in soil. The hydrolysis half-life in soil is >13 to 240 days (Howard et al., 1991). Under aerobic conditions, the half-life of lindane is 31 to 413 days; whereas under anaerobic conditions, the half-life is 6 to 30 days. Lindane can reach the atmosphere by wind erosion of soil particles to which it is sorbed and through volatilization. Once in the atmosphere, lindane does not appear to undergo significant photodegradation or other degradation processes. The photo-oxidation half-life of lindane in air is about 3 days (Howard et al., 1991). The loss of lindane from the atmosphere is caused primarily by rain washout and dry deposition. Lindane that is released to water has a tendency to dissolve and remain in the water column. It is about 50,000 times more soluble in water than DDT. Evaporative loss from water does not appear to be an important migration pathway. In water, lindane may undergo hydrolysis and adsorption/desorption processes with sediments and other materials. Partitioning to aquatic organisms to higher levels than the surrounding water concentrations may also occur. However, lindane and other isomers of BHC do not appear to undergo biomagnification to a great extent.

### 5.1.3 Benzene

The high volatility and fairly high solubility of benzene are two properties that have the greatest influence on the environmental fate of benzene. When deposited to soils or released to water, substantial volatilization of benzene is likely to occur. Once volatilized, atmospheric benzene undergoes significant chemical degradation, primarily by reacting with hydroxy radicals. Some photo-oxidation may also occur, with a half-life of 2 to 21 days (Howard et al., 1991). The photo-oxidation half-life in water is much

longer, 334 days to >36 years. Because of its solubility in water and weak adsorption to soil particles, benzene is expected to be fairly mobile between soil and water. Benzene is not susceptible to hydrolysis but may undergo biodegradation by aerobic or anaerobic micro-organisms. Under anaerobic conditions, benzene may persist in soils for months to years; under aerobic conditions, the half-life of benzene in soil and water is 5 to 16 days. Benzene is not expected to accumulate in aquatic organisms.

#### **5.1.4 Chlorobenzene**

For chlorobenzene, chemical and physical properties such as soil adsorption coefficient and bioconcentration factor (BCF) indicate that it would be moderately adsorbed to soil particles and have little or no tendency to bioaccumulate. Chlorobenzene's sorption on soil particles increases with increasing soil organic content; it may also volatilize from soil surfaces. Hydrolysis is not expected to be a significant transformation process, although chlorobenzene may be biodegraded. Under aerobic and anaerobic aqueous conditions, its half-life is 68 to 150 days and 272 to 600 days, respectively (Howard et al., 1991). Chlorobenzene is persistent in water with a half-life of 68 to 150 days. Evaporation is the major route of migration from water. Air is an important medium for the transport and transformation of chlorobenzene. Once in the atmosphere, the photo-oxidation half-life of this chemical is 3 to >30 days, whereas photo-oxidation in water may take months to years.

#### **5.1.5 Chloroform**

Chloroform weakly adsorbs to soil and is highly mobile in aqueous systems. Hydrolysis is significant in natural soils, but some biodegradation of chloroform may occur. Under anaerobic conditions, the half-life is 1 to 4 weeks, whereas the half-life under aerobic conditions is 4 weeks to 6 months (Howard et al., 1991). Sorption of chloroform on soil particles is expected to increase with increasing soil organic matter content. Chloroform on the soil surface is likely to rapidly volatilize. Diffusion of chloroform

through the soil-air pores up to the ground surface, and subsequent removal by wind, may be a significant loss pathway in unsaturated soils. If chloroform enters surface waters, it will be lost primarily through evaporation. The primary reaction of vapor-phase chloroform is with photochemically generated hydroxyl radicals in the atmosphere. The half-life by this degradation process is estimated to be 70 to 79 days (Howard, et al., 1991). Once in the atmosphere, the photo-oxidation half-life is 26 to 260 days (Howard et al., 1991). In most cases, it should be assumed that chloroform will persist for months to years (BEIA, 1989). However, chloroform has no significant potential for bioaccumulation.

#### **5.1.6 1,2-Dichloroethane**

The chemical and physical properties of 1,2-dichloroethane (1,2-DCA) indicate that it is expected to be highly volatile, weakly adsorbed to soils, and soluble in water. It is likely to move readily into groundwater. Sorption on soil particles is expected to increase with increasing soil organic matter content. Biological degradation in natural soil and water systems is not expected to be significant. The half-life of 1,2-DCA under anaerobic conditions is 400 days to 2 years, and the half-life under aerobic conditions is 100 days to 6 months (Howard et al., 1991). In most cases, 1,2-DCA is expected to persist for months to years. The principal removal route in surface waters is evaporation. The half-life of 1,2-DCA in air is 12 to 122 days.

#### **5.1.7 Ethylbenzene**

Ethylbenzene is expected to be moderately adsorbed to soils, to volatilize from soil surfaces, and to be fairly mobile in soil. Volatilization losses through air-filled pores may be a minor loss pathway. This chemical is resistant to hydrolysis but readily undergoes biodegradation if microbial populations are sufficiently numerous and active. Under aerobic conditions, the half-life is 3 to 10 days; under anaerobic conditions, the half-life is 176 to 228 days (Howard et al., 1991). It may persist for months to years if

biodegradation is not possible. Ethylbenzene in surface water will vaporize to a large extent or biodegrade. An estimate of ethylbenzene's half-life in water is 3 to 10 days; its half-life in air is approximately 3 days. Ethylbenzene does not have a significant bioaccumulation potential.

### **5.1.8 Toluene**

Based upon its chemical and physical properties (Table 5-1), toluene is expected to be moderately adsorbed to soil particles and to be relatively mobile in aqueous systems. This compound is resistant to hydrolysis but may undergo biodegradation if microbiological populations are sufficiently numerous and active. When released to surface waters, toluene will tend to vaporize; that which remains will be subject to biodegradation. Under aerobic and anaerobic conditions, its half-life is 4 to 22 days and 8 to 30 weeks, respectively. The bioaccumulation potential of toluene is moderate. Toluene in the atmosphere is degraded by reaction with hydroxyl radicals. The half-life of toluene in air is approximately 4 days (Howard et al., 1991).

### **5.1.9 Xylenes**

Xylene isomers are expected to be highly volatile from aqueous solutions, moderately adsorbed to soil particles, and relatively mobile in aqueous systems. Volatilization is the dominant transport mechanism of xylene. Xylenes can volatilize from surface soil through air-filled pores. Once in the air, the estimated half-life is about 1 to 2 days (Howard et al., 1991). Xylenes are resistant to hydrolysis but may undergo biodegradation. Under aerobic conditions, the estimated half-life of xylenes is 1 to 4 weeks; under anaerobic conditions, the estimated half-life is 6 to 12 months. It is likely that xylenes persist in the soil for months to years and have some tendency to bioaccumulate (BEIA, 1989).

## 5.2 Exposure Pathways

An exposure pathway describes how a contaminant may move from its source to a receptor (a potentially exposed organism). A complete exposure pathway has five primary elements:

- A chemical source
- A mechanism of release
- An environmental medium
- An exposure point (receptor location)
- A feasible route of exposure (e.g., ingestion)

An exposure pathway is complete if there is a reasonable likelihood that a receptor may take in contaminants through contact with contaminated media. No exposure (and thus no risk) exists unless the exposure pathway is complete. A schematic diagram of the potential exposure pathways to ecological receptors in the Montrose study area is presented on Figure 5-1.

The primary source of Montrose-related contaminants was the central process area, with releases occurring during normal operations (manufacturing/processing, storage, and shipment) and probably also during the demolition of the facility. This risk assessment describes the potential effects of the release of chemicals from the Montrose property through surface drainage (aquatic pathways) and atmospheric transport (mainly terrestrial pathways). Releases of chemicals of concern through the sanitary sewer system (and the associated effects) are being addressed separately, as described earlier.

Actual effects of the release of chemicals through these pathways have not been studied thoroughly. However, available information is summarized in Section 6.4.

### 5.2.1 Aquatic Pathways

After release from the Montrose property, contaminants may have migrated or been transported through the aquatic pathway by various media, including surface water, sediment, and groundwater. Among those, surface water and sediment are the most significant within the study area; groundwater transport would be ecologically significant if the chemicals were water-soluble and had a low to moderate affinity for soils, and if the groundwater discharged to the surface with the chemicals still in solution. However, groundwater transport from the Montrose property does not appear to be a significant source for ecological receptor exposure because groundwater does not discharge to the surface (see Section 2.5).

Concentrations of Montrose-related chemicals have been measured in surface water and sediment from the Torrance Lateral, Dominguez Channel, and Consolidated Slip (see Section 3). These data indicate that DDT, DDE, DDD, and BHC isomers, including lindane, were transported through the surface drainage system and that these chemicals may be redistributed by re-suspension and movement of the sediments with which they are associated. Several of the contaminants of ecological concern (especially DDT, DDE, and DDD) have a high affinity for organic-rich sediments or soils and would tend to move only slowly through the soil; however, sediment-sorbed contaminants would be transported readily through erosion of sediments or soils.

The exposure points of greatest ecological concern within the aquatic pathway include the mouth of the Torrance Lateral (near the San Diego Freeway where this channel does not have a concrete lining), the Dominguez Channel, and the Consolidated Slip. Aquatic and semi-aquatic organisms occur in those areas and may be exposed to Montrose-related chemicals that have been transported with surface drainage (see Section 4, Ecological Receptors). Portions of the drainage system between the site and the confluence of the Torrance Lateral with the Dominguez Channel appear to be less significant ecologically because that drainage is through underground storm drains or

concrete-lined channels that do not provide important habitats for ecological receptors. However, contaminated sediments in underground drains (e.g., Kenwood Drain) would be of concern if they served as a reservoir for continued discharge of contaminants (H+A, 1990).

The aquatic and terrestrial organisms in the Montrose study area may be exposed to contaminants through various mechanisms (such as ingestion, dermal contact, or inhalation) that could result in metabolic uptake or absorption. Chemical and physiological processes are also involved because uptake by the three mechanisms mentioned does not necessarily mean the contaminant will be incorporated into living tissue or be toxic for two primary reasons: (1) the amount that actually reaches the systemic circulation depends on bioavailability of the compound, and (2) some contaminants may be ingested but then metabolized to a nontoxic form and excreted. Other contaminants or their metabolites may be toxic and remain in the body for long periods of time. Absorption, metabolism, excretion—and hence toxicity—may all be dose dependant.

Ingestion of contaminated surface water or sediment by aquatic or semi-aquatic organisms is probably a principal uptake mechanism, along with direct contact with those media. Such ingestion or direct contact would be most significant for benthic invertebrates inhabiting contaminated portions of the drainage system. Benthic macroinvertebrates (such as polychaetes and crustaceans) living in or on contaminated sediments are directly exposed to contaminants in the substrate. Many aquatic organisms (including benthic invertebrates and fish) take in some contaminants by absorption across gill membranes and integument (e.g., skin) in addition to exposure through ingestion. Therefore, those organisms are continually exposed to the contaminants, and uptake rates may be governed mostly by the characteristics of the contaminant (i.e., a passive chemical and physical mechanism). Semi-aquatic birds (such as waterfowl and shorebirds) may ingest substantial amounts of sediments along with their prey when feeding (Beyer et al., 1992). For example, sandpipers, which probe or peck for invertebrates in the mud or shallow water, may consume soil at a rate of up to 30 percent of their diet. Most of the mallards in the study by Beyer et al. (1992) contained little or no sediment;

however, the 10 percent with the most sediment had consumed an estimated 16 percent sediment in their diet. Inhalation may be an important exposure mechanism for semi-aquatic birds if highly volatile toxic chemicals are present in the water.

Available information indicates that aquatic and semi-aquatic animals are present in the surface water bodies of the study area and there are potentially complete exposure pathways from the primary source (Montrose property) to these organisms.

### **5.2.2 Terrestrial Pathways**

After release from the property, contaminants may have migrated or been transported through the terrestrial pathway by soil/dust or air. The relative significance of those media varies by chemical. For example, the more volatile chemicals (such as benzene and chloroform) would have been transported largely by air, whereas the volatile chemicals that also adsorb readily to soil particles (such as DDT, DDE, and DDD) would have been transported by soil/dust as well as air. The volatile chemicals can spread quickly over relatively long distances through the air, but in the process they are greatly diluted.

Some DDT has moved from the Montrose property to downwind areas. Surface soils to the southeast contained up to 7.6 mg/kg of DDT within 1 mile from the site. Within 0.5 miles from the site, concentrations detected were as high as 250 mg/kg (Figure 3-17). Terrestrial habitats in downwind areas could be significant exposure points if contaminated soil/dust were deposited there historically. Much of the downwind area has been subjected to commercial/industrial and residential development, but some habitats in the downwind areas are used by terrestrial wildlife and their associated food organisms (such as earthworms and insects).

Terrestrial organisms in the downwind areas could be exposed to contaminants through ingestion, dermal contact, or inhalation, which could result in metabolic uptake. If

burrowing and ground-dwelling mammals (such as voles, pocket gophers, and mice) and invertebrates (such as earthworms and insects) ingest contaminated soils or inhale contaminant vapors, exposure pathways would be complete. Earthworms and other invertebrates can accumulate persistent soilborne insecticides and are a significant source of contamination of terrestrial wildlife (EPA, 1975; Beyer and Gish, 1980; Beyer and Krynitsky, 1989; Beyer, 1990).

### **5.2.3 Food Chain Relationships**

All the contaminants of ecological concern (Table 3-9) are toxic to living organisms under certain exposure conditions (see Section 6, Toxicity Assessment). Although acute toxicity may be more important for some primary receptors, chronic toxicity is probably more important for the contaminants of greatest ecological concern (e.g., DDT and metabolites), especially for the secondary receptors. In addition to direct toxic effects, those organisms could be affected through habitat degradation resulting from acute or chronic toxicity to the lower trophic levels of the food chain.

There are two major aquatic food chain exposure pathways for Montrose-associated contaminants. First, the chemicals may be in dissolved form or associated with suspended particles in the water column of the Dominguez Channel or Consolidated Slip. In this case, aquatic exposure would occur primarily through the planktonic and nektonic food webs and eventually affect aquatic birds. Exposure could also occur through those plants and animals living on the surface of hard and soft channel substrates. In the second type of pathway, contaminants would be adsorbed to sediments or dissolved in the interstitial water of accumulated sediments at the bottom of the channels. In that case, the possibility exists that contaminants may be deeply buried, commonly exposed to anaerobic conditions, and/or (as a result) partially or completely isolated from biotic exposure. Although the two pathways contain different types of organisms, they occur in physical proximity and are closely linked ecologically. Food web linkages for Los Angeles Harbor, representative of the Consolidated Slip and sharing many species

in common with Dominguez Channel, are shown on Figure 5-2. These food web linkages represent exposure pathways beginning with sediment and water column uptake of chemicals of concern.

Food chain relationships throughout the study area have not been investigated in detail. However, based on the reconnaissance-level surveys and other available information, the most susceptible receptors are probably those animals that live in or on potentially contaminated sediments in the lower Torrance Lateral, Dominguez Channel, and Consolidated Slip, or higher trophic level consumers that feed on those organisms. Examples of those consumer species include fish, waterfowl, shorebirds, cormorants, grebes, and other birds. While remaining in the study area, the more sedentary species (at least seasonally) are likely to receive the highest exposures, particularly to the chemicals such as DDT, DDE, and DDD that biomagnify through the food chain.

Although few studies of the food chain relationships have been conducted in the study area, the results of studies conducted there or elsewhere indicate that those chemicals could be expected to occur in fish or birds at thousands of times the concentration found in their foods and that dietary concentrations of a few mg/kg can affect survival or reproduction (see Section 6, Toxicity Assessment). In aquatic habitats of the study area, probable food chains include fish feeding on invertebrates (which may have fed on other invertebrates) and birds feeding on those fish or invertebrates. In the terrestrial habitats, birds and mammals may feed on earthworms or other invertebrates that have accumulated contaminants from the soil.

No sampling results are available for food chain organisms collected near the Montrose property other than those from the State Mussel Watch (see Section 6.4, Known Effects in the Study Area). Toxicity tests and population studies other than those associated with dredging projects have not been conducted in the study area. Results of the dredging project studies also are summarized in Section 6.4.

The Dominguez Channel and Consolidated Slip are slow-moving, marine/estuarine environments generally supportive of an open-water community and characteristic of southern California harbors (Soule and Oguri, 1980). The community is composed of a complex assemblage of plant and animal plankton and fish and invertebrate nekton (larger, open-water swimming organisms). Aquatic birds such as grebes, cormorants, herons, mergansers, and gulls enter this exposure pathway by preying on fish. It is characteristic of the open-water community that biomass is produced through phytoplankton and bacterial production, which serves to trap and temporarily suspend chemicals within the biota and on the surfaces of detritus in the upper water column. It is equally important to note that the open-water community strips chemicals from the water by transporting biotic-incorporated contaminants to the sediment as part of a continuous rain of settling detritus. Contaminants are initially incorporated into the community through external integument exposure and adsorption or feeding on contaminated particles at the microbial or phytoplankton level. The contaminants undergo varying degrees of bioaccumulation dependent on chemical-specific and organism-specific characteristics. See Section 6-3, Bioaccumulation Potential, for additional information on contaminant accumulation in these environments.

DDT in the Montrose drainage system appears to be trapped in drainage sediments (e.g., Dominguez Channel sediments) (H+A, 1990; Envirologic Data, Inc., 1991). Surface water contamination is likely associated with resuspended sediment during wet-weather flows. During low flow, dry-weather periods, most organic contaminants are trapped in the sediments where direct exposure is limited to burrowing benthic organisms. Polychaetes and amphipods (Tables 4-1 and 4-4) are common benthic invertebrates of the Dominguez Channel and Consolidated Slip that are available to birds and fish at the shallow sediment-accumulation areas at the Torrance Lateral/Dominguez Channel junction and the harbor end of the Consolidated Slip. Fish may consume these substrate-associated benthic invertebrates at any water depth in the Channel or Slip. The consumption of benthic organisms by fish and birds is a likely route for moving contaminants from the substrate up into the rest of the food web.

Figure 5-2 shows the trophic links between the benthic, planktonic, and nektonic food webs and likely routes of contaminant exposure to aquatic birds.

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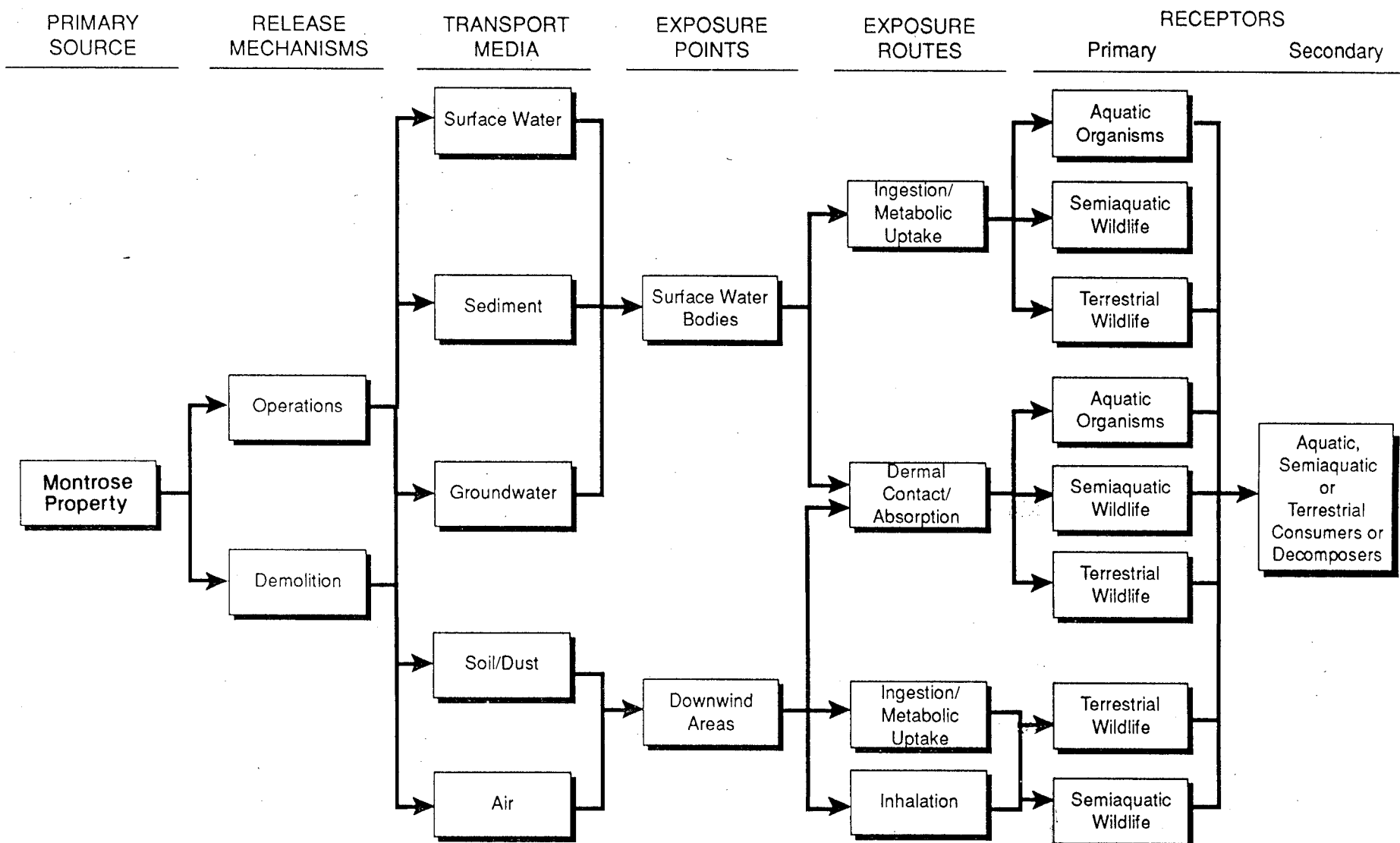
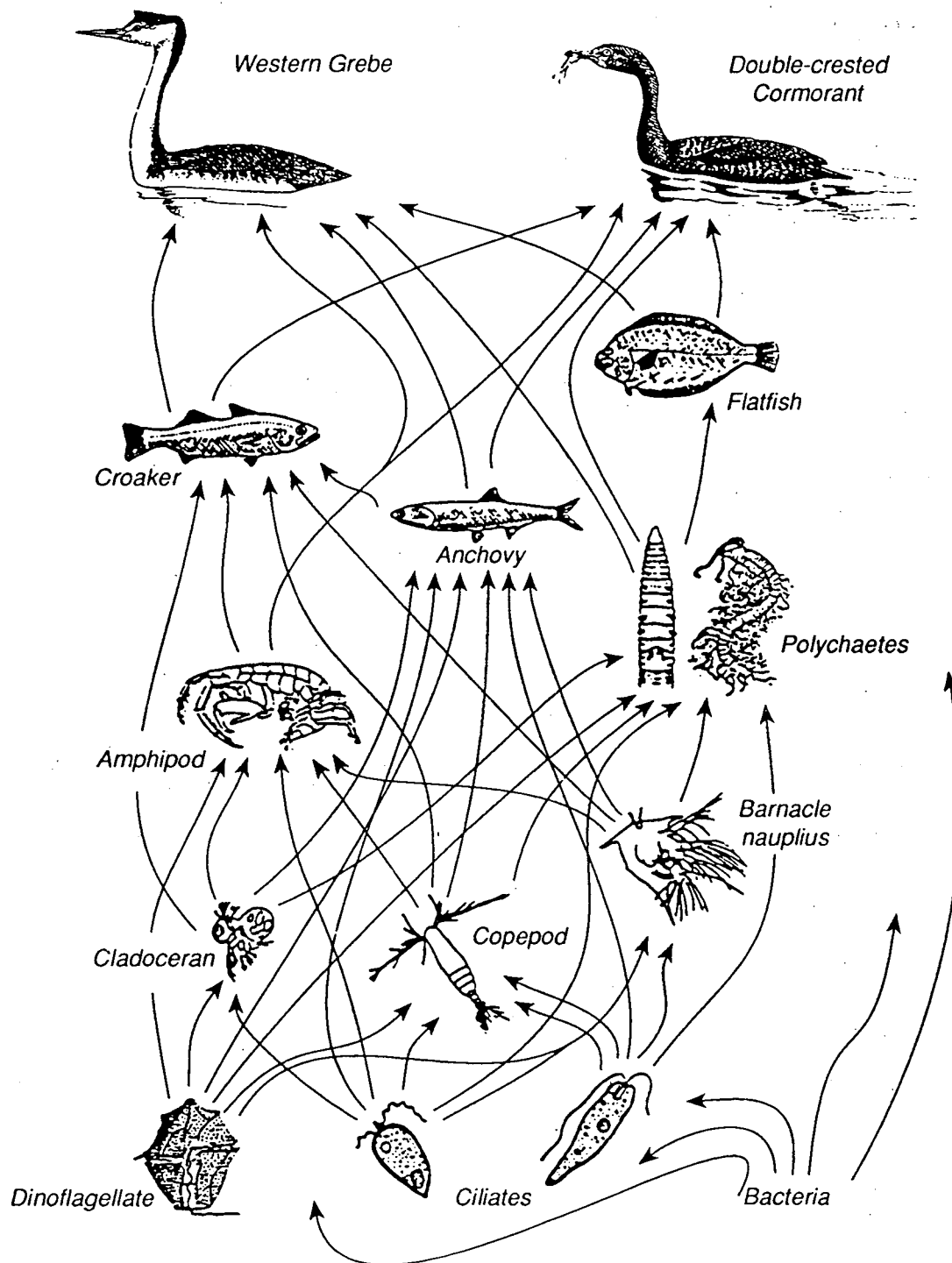


FIGURE 5-1  
POTENTIAL EXPOSURE PATHWAYS  
FOR ECOLOGICAL RECEPTORS

Ecological Risk Assessment  
Montrose Superfund Site





Source: Modified from Soule and Oguri, 1980

FIGURE 5-2  
POTENTIAL FOOD CHAIN  
RELATIONSHIPS IN AQUATIC HABITATS

Ecological Risk Assessment  
Montrose Superfund Site

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## **6 Toxicity Assessment**

## Section 6

### Toxicity Assessment

#### 6.1 Criteria

##### 6.1.1 Ambient Water Quality Criteria and Sediment Criteria

Criteria developed under the State of California Bays and Estuaries Plan (SWRCB, 1992) and EPA Water Quality Criteria (EPA, 1986) are summarized in Table 6-1 for the Montrose chemicals of concern. Suggested sediment criteria being developed by EPA are shown in Table 6-2. These acute and chronic exposure levels can be used as a gauge to judge present levels of contamination observed in the Montrose drainage system. Note that there are neither California nor EPA marine water quality criteria for several chemicals because insufficient data exist to establish criteria for the compounds.

##### 6.1.2 No Observed Adverse-Effect Levels (NOAEL) and Lowest Observed Adverse Effect Levels (LOAEL)

Effect levels of exposure vary widely among species and methods of toxicant exposure for the contaminants of concern (Appendix B). For example, mammals and some species of birds (especially the gallinaceous species such as chickens and quail) are much less sensitive to the effects of DDT and its metabolites than some other birds (such as waterfowl, raptors, and pelicans [see also Section 6.2]). The effect levels also vary according to the body condition, age, sex, or reproductive condition of test animals.

Lowest observed adverse-effect levels for saltwater aquatic life are summarized in Table 6-3.

**Table 6-1**  
**Water Quality Criteria for Contaminants of Concern**

Compound	California Bays and Estuaries Plan		EPA	
	Daily Average	30-Day Average <sup>a</sup>	Marine Acute	Marine Chronic
DDT-total (ng/L)	1.0	0.6	130	1.0
DDE-total				
DDD-total				
BHC-total				
gamma-BHC (Lindane) (ng/L)	160	62	160	
Benzene (µg/L)		21		
Chlorobenzene (mg/L)		4.5		
Chloroform (µg/L)		480		
1,2-Dichloroethane (µg/L)		130		
Ethylbenzene (mg/L)		29		
Toluene (mg/L)		300		
Xylene				
<sup>a</sup> Human-health standard; state objectives for aquatic life established only for daily average DDT and lindane  Blanks indicate criteria are not available.  Source: SWRCB, 1992 and EPA, 1986				

**Table 6-2**  
**Suggested Sediment Criteria**

Compound	Mean Criteria <sup>a</sup> ( $\mu\text{g/gC}$ )	Sediment "Safe" Level <sup>b</sup> ( $\mu\text{g/g-OC}$ )	
		Acute	Chronic
DDT	0.828	21	0.16
DDD		325	
DDE		700	
Gamma-BHC		0.31	
Benzene		245	34
Ethylbenzene		140	
Toluene		315	250

<sup>a</sup>EPA, 1988. Interim Sediment Criteria Values for Nonpolar Hydrophobic Organic Contaminants.

<sup>b</sup>Pavlou, 1987. The Use of Equilibrium Partitioning Approach in Determining Safe Levels of Contaminants in Marine Sediments.

Blank spaces mean no criteria are available.

**Table 6-3**  
**Lowest Observed Adverse Effect Levels (LOAEL)**  
**( $\mu\text{g/L}$ ) for Saltwater Aquatic Life**

Compound	Acute	Chronic
DDT-Total	<sup>a</sup>	<sup>a</sup>
DDE-Total	14	
DDD-Total	3.6	
BHC-Total	0.34 <sup>b</sup>	
Gamma-BHC (Lindane)	<sup>c</sup>	<sup>c</sup>
Benzene	5100	700 <sup>d</sup>
Chlorobenzene	160	129
Chloroform		
1,2-Dichloroethane	113,000	
Ethylbenzene	430	
Toluene	6300	5000
Xylene		

<sup>a</sup>For DDT and its metabolites, the criterion to protect saltwater aquatic life is 0.0010  $\mu\text{g/L}$  as a 24-h average and the concentration should not exceed 0.13  $\mu\text{g/L}$  at any time.

<sup>b</sup>Acute toxicity occurs at concentrations as low as 0.34  $\mu\text{g/L}$  BHC.

<sup>c</sup>The concentration of lindane should not exceed 0.16  $\mu\text{g/L}$  at any time.

<sup>d</sup>Adverse effects occur at concentrations as low as 700  $\mu\text{g/L}$  with a fish species exposed to benzene for 168 d.

Source: BEIA, 1989

As can be seen from Tables 6-1 and 6-3 and Appendix B, the Acute Lowest Observed Adverse-Effect Levels (LOAEL) and criteria for protection of saltwater aquatic life range from 0.13  $\mu\text{g/L}$  for DDT to 113 mg/L for 1,2-dichloroethane. These data are a result of laboratory tests conducted under controlled conditions with lethality as the test endpoint. In general, the pesticide compounds DDT (and its metabolites DDE and DDD) and BHC (and its isomers) are more acutely toxic to saltwater aquatic life than the aromatic and chlorinated hydrocarbon compounds (Table 6-3). There are few laboratory studies that report chronic LOAEL for saltwater aquatic life. As shown in Tables 6-1 and 6-3, chronic, nonlethal effects concentrations and aquatic life criteria for the contaminants of concern range from 0.001  $\mu\text{g/L}$  for DDT to 5,000  $\mu\text{g/L}$  for toluene.

The low concentration of DDT is based on bioaccumulation in the foodchain, especially in fish such as northern anchovies that are eaten by brown pelicans in Southern California. Total DDT concentrations of 150  $\mu\text{g/kg}$  in these anchovies have been associated with improved reproduction in brown pelicans following reduction of DDT discharges in the 1970s from the Montrose property through the LA County sewer system (Anderson et al., 1975). However, with total DDT concentration at 150  $\mu\text{g/kg}$  in anchovies, reproduction still was 10 to 30 percent below those levels needed to maintain a stable population (Anderson et al., 1977).

### **6.1.3 Effects Range-Low (ER-L) and Effects Range-Median (ER-M) in Sediments**

The National Oceanic and Atmospheric Administration (NOAA) annually collects and analyzes sediment samples from sites located in coastal marine and estuarine environments throughout the United States as a part of the National Status and Trends (NS&T) Program (Long and Morgan, 1990). The chemical data generated from this program provide an indication of the relative degrees of contamination of sediments, but do not provide a measure of the adverse biological effects or an estimate of the potential for such effects. Thus, data associated with biological effects from a wide

variety of studies were assembled and evaluated by NOAA (Long and Morgan, 1990). The data from three basic approaches to establishing effects-based criteria were evaluated: (1) the equilibrium-partitioning approach, (2) the spiked-sediment bioassay approach, and (3) various methods of evaluating synoptically collected (broad-scale survey) biological and chemical data in field surveys. The chemical concentrations observed or predicted by the different methods to be associated with biological effects were sorted, and the lower 10 percentile and median concentrations were identified along with an overall apparent effects threshold. The lower 10 percentile in the data was identified as an Effects Range-Low (ER-L) and the median as an Effects Range-Median (ER-M) concentration for each evaluated chemical. These NOAA ER-L and ER-M concentrations are not carbon normalized (i.e., they are not related to total organic carbon content of sediments). They are, therefore, of limited usefulness when comparing to field data and are not to be construed as NOAA standards or criteria.

The ER-L values are concentrations equivalent to the lower 10 percentile of the screened available data and indicate the low end of the range of concentrations at which effects were observed or predicted. The ER-L identifies the concentrations above which adverse effects may begin or are predicted among sensitive life stages and/or species or as determined in sublethal tests.

The ER-M values are the concentrations equivalent to the 50 percentile point in the screened available data. The ER-M identifies the concentrations above which effects were frequently or always observed or predicted among test species.

Table 6-4 presents the ER-L and ER-M values for DDT, DDE, and DDD as determined by the NS&T Program (Long and Morgan, 1990). There were insufficient data to determine ER-L and ER-M values for other contaminants of ecological concern.

**Table 6-4**  
**Effects Range-Low and Effects Range-Median ( $\mu\text{g/kg}$ ) as Determined by**  
**the National Status and Trends Program**

Compound	Effects Levels	
	ER-L	ER-M
p,p'-DDT	1.0	7.0
p,p'-DDE	2.0	15.0
p,p'-DDD	2.0	20.0
Total DDT	3.0	350.0
<p>Note: Data for other contaminants of concern are insufficient for identification of ER-L and ER-M concentrations. See text for definitions of ER-L and ER-M.</p> <p>Source: Long and Morgan, 1990</p>		

The ER-L value for p,p'-DDT of 1.0  $\mu\text{g/kg}$  is supported by sediment-water equilibrium partitioning-based thresholds of 0.7 and 1.6  $\mu\text{g/kg}$  (assuming 1 percent total organic carbon content) (Long and Morgan, 1990). The ER-M value of 7.0  $\mu\text{g/kg}$  for p,p'-DDT is supported by moderate toxicity to bivalve larvae (6.6  $\mu\text{g/kg}$ ) and significant toxicity to amphipods (7.5  $\mu\text{g/kg}$ ) exposed to San Francisco Bay sediments. With several exceptions, effects were usually observed at concentrations of about 6  $\mu\text{g/kg}$  or greater. The degree of confidence in the p,p'-DDT ER-L and ER-M values should be considered as low. This low confidence may be because the values are not based on organic carbon content in sediment, especially given the affinity of DDT for organic carbon.

The ER-L value of 2  $\mu\text{g/kg}$  for p,p'-DDE is supported by apparent effects threshold-based data and bioassay data from San Francisco Bay sediments tested with *Rhepoxynius abronius* amphipods and bivalve larvae (2.2, 2.2, 2.1, 2.2  $\mu\text{g/kg}$ ) (Long and Morgan, 1990). Effects were almost always seen in association with concentrations exceeding 2  $\mu\text{g/kg}$ . The ER-M value of 15  $\mu\text{g/kg}$  for p,p'-DDE is supported by relatively few data points. An apparent effects threshold could not be determined because of the lack of sufficient data. The degree of confidence in the p,p'-DDE ER-L and ER-M values should be considered as moderate and low, respectively, for reasons similar to those given for DDT values.

The ER-L and ER-M values for p,p'-DDD (2 and 20  $\mu\text{g/kg}$ , respectively) are supported by apparent effects threshold-based data from Puget Sound (2 and 16  $\mu\text{g/kg}$ , respectively) (Long and Morgan, 1990). There were too few data to justify identifying an apparent effects threshold. The degree of confidence for both ER-L and ER-M values for p,p'-DDD should be considered as low for reasons similar to the limited confidence in the DDT values.

The ER-L value of 3  $\mu\text{g/kg}$  for total DDT is supported by only two sediment-water equilibrium partitioning-based thresholds (1.58 and 3.29  $\mu\text{g/kg}$ ) and one freshwater screening level concentration (1.9  $\mu\text{g/kg}$ ) (Long and Morgan, 1990). The ER-M value of 350  $\mu\text{g/kg}$  for total DDT is supported by observations of moderate abundances of arthropods in southern California sediments (mean 350  $\mu\text{g/kg}$ ) and low taxa richness in DuPage River macrobenthos (mean 222  $\mu\text{g/kg}$ ). Series of spiked-sediment assays with *Hyaella azteca* demonstrate the importance of organic carbon in regulating bioavailability, and, therefore, toxicity of sediment-associated DDT. There was no overall apparent threshold in concentration of total DDT above which effects were usually or always observed. The degree of confidence in the ER-L and ER-M values for total DDT should be considered as moderate.

There were insufficient data to determine ER-L and ER-M values for lindane or other contaminants of concern in the Montrose study area (Long and Morgan, 1990). Most

of the samples were not tested for lindane or had nondetectable concentrations. Using the sediment-water equilibrium partitioning-based approach, effects would be predicted to occur at concentrations ranging from 1.57 to 12  $\mu\text{g/kg}$  dry weight.

## **6.2 Toxic Endpoints**

The contaminants of concern have the potential to cause toxic effects in laboratory and wild animals. Toxicity depends on dose, route of exposure, duration of exposure, and the ability of the body to metabolize and eliminate the toxicant. Available information concerning no-effects, sublethal and chronic effects, and acute effects of the contaminants of concern is summarized in Appendix B.

The following section provides a brief summary of the modes of action and toxicity of each contaminant. Emphasis is placed on wild aquatic, semi-aquatic, and terrestrial species that may be found in the study area or that are related to species in the study area.

### **6.2.1 DDT**

DDT is a neurotoxicant that acts primarily on the central nervous system (CNS), changing the transport of sodium and potassium across nerve axon membranes. The transmission of nerve impulses is interrupted and hyperpolarization of the axon is produced (Hodgson and Levi, 1987). Single large doses or repeated doses can produce hyperexcitability, tremors, ataxia, and epileptiform convulsions (BEIA, 1989). Death from DDT is usually the result of respiratory arrest.

In mammals, DDT is metabolized by two pathways. It is converted to a slight extent to DDE, which does not undergo further biotransformation, but is stored indefinitely in adipose tissues. The major detoxification pathway is via dechlorination to DDD, which

is readily degraded to a water-soluble metabolite, DDA, and is rapidly excreted into the urine (WHO, 1979). Bailey et al. (1969a, b) determined a half-life of elimination of DDT in pigeons of 28 days.

Age and size are two important factors influencing susceptibility to DDT poisoning. This is very much evident in fish, for which insecticide toxicity has been assessed by means of concentration changes in the ambient water (not the amount given per unit body weight, as with other animals). For example, relatively low concentrations can affect the hatchery spawning and rearing operations for coho salmon (*Oncorhynchus kisutch*). Laboratory studies of DDT toxicity to various stages and sizes of coho salmon have confirmed that older fish are more resistant to DDT (Matsumura, 1985). This is probably related to lower metabolic rates in older and larger fish as contrasted to younger and more rapidly growing larval and juvenile fish.

#### **6.2.1.1 Acute Effects**

The acute toxicity of technical DDT appears to be caused almost exclusively by the p,p'- DDT isomer. The oral LD<sub>50</sub> (lethal dose) in rats and rabbits is 87 and 250 mg/kg, respectively (RTECS, 1984). Hudson et al. (1984) determined the acute toxicity (expressed as LD<sub>50</sub>) of orally administered DDT to several wildlife species:

- Female bullfrog (*Rana catesbeiana*), >2,000 mg/kg
- Female mallard duck (age 3 months), >2,240 mg/kg
- Male California quail (*Callipepla californica*) (age 6 months), 595 mg/kg
- Male Japanese quail (age 2 months), 841 mg/kg
- Female pheasant (*Phasianus colchicus*) (age 3 to 4 months), 1,334 mg/kg
- Adult male and female sandhill cranes (*Grus canadensis*), >1,200 mg/kg
- Male and female rock doves (*Columba livia*), >4,000 mg/kg

Signs of intoxication included ataxia, wing-drop, jerkiness in gait, continuous whole-body tremors, falling, and convulsions.

In acute laboratory studies with saltwater fish, DDT 48-hour  $LC_{50}$  concentrations of 0.4 and 1.8  $\mu\text{g/L}$  were determined for spot (*Leiostomus xanthurus*) and striped mullet (*Mugil cephalus*) respectively (EPA, 1980a). Acute  $LC_{50}$ s of between 0.63  $\mu\text{g/L}$  and 0.83  $\mu\text{g/L}$  were determined for the saltwater sand shrimp (*Crangon septemspinosa*) (McLeese and Metcalfe, 1980).

#### 6.2.1.2 Sub-Acute and Chronic Effects

DDT has been reported to exhibit estrogenic properties following in vivo administration to a number of mammalian species (BEIA, 1989). The estrogenic action of technical DDT resides in the o,p'-isomer. Its estrogenic activity is about one-ten thousandth of that of estradiol. Female rats exposed to 0.1 mg o,p'-DDT (no route specified) on the second, third, or fourth days of life showed precocious puberty, persistent vaginal estrus, and anovulation (Gellert et al., 1974). Male neonate rats injected with 3 mg of o,p'-DDT 1 to 3 hours after birth showed an abnormal pattern of sexual brain differentiation that was attributed to inhibition of the normal action of testosterone on the developing brain (Lee and Visek, 1975).

DDT exhibits estrogenic properties in gulls (*Larus* sp.). In two studies (Fry and Toone, 1981; Fry et al., 1987), injection of DDT into gull eggs at concentrations comparable to those found in contaminated seabird eggs in southern California in the 1970s resulted in abnormal development of both male and female embryos. DDT caused feminization in males with characteristic localization of germ cells in a thickened ovary-like cortex. The reproductive tract of females was affected by DDT, with characteristic persistence of the right oviduct, which is abnormal. The most estrogenic DDT isomer was o,p'-DDT.

In animals given repeated doses of DDT, pathological lesions are seen in the liver and kidneys (Klaassen et al., 1986). Histopathological changes were observed in the livers of rats exposed to dietary levels as low as 5 mg/kg for 4 to 6 months (NIOSH, 1978).

Wiemeyer et al. (1986) fed captive American kestrels (*Falco sparverius*) long-term dietary dosages of DDT and dieldrin in combination to compare reproductive success and eggshell thickness with that of controls. High-treatment birds received a diet containing 15 mg/kg DDT and 3 mg/kg dieldrin (dry-weight). Low-treatment birds received a diet containing 5 mg/kg DDT and 1 mg/kg dieldrin (dry-weight). Heavy mortality involving only dosed birds occurred during periods of temperature declines and other stress factors. The eggshells of dosed groups were 6 to 17 percent thinner than those of controls. The shell thickness of offspring maintained on the same dosage as their parents, including those placed on clean food before egg laying, was 12 to 23 percent less than that of controls. Reproductive success of dosed birds was clearly related to contaminant concentrations in a sample egg from each pair. The percent of young fledged and the number of young fledged per pair was depressed when contaminant concentrations were high. Poor productivity was caused primarily by reductions in hatching success through egg disappearance and mortality of nestlings.

Coturnix quail were used in a study by Gish and Chura (1970) to evaluate the effects of dietary DDT under simulated conditions of wild birds during breeding and migration. Light conditions were manipulated to stimulate or suppress reproductive development, and some birds were partially starved or fully fed before dosage. Birds were then fed dietary levels of 0, 700, 922, 1,214, or 1,600 mg/kg (dry weight) of DDT for a period of 20 days or until death. Partially starved birds were more susceptible to DDT intoxication than fully fed ones, and males died earlier than females. Females in breeding condition were less sensitive to DDT than were nonbreeding females and males. After 10 days on dosage, however, the cumulative mortality of females in breeding condition rapidly approached that of males and of females not in breeding condition.

The onset of spring migratory condition was delayed at least 1 week in caged white-throated sparrows (*Zonotrichia albicollis*) fed diets containing 5 or 25 mg/kg technical DDT (Mahoney, 1975).

Increased respiratory rates in juvenile blue crabs (*Callinectes sapidus*) were demonstrated at a concentration of 800  $\mu\text{g/L}$  in sublethal toxicity studies conducted with DDT exposures in food (Leffler, 1975, as cited by Ballou et al., 1985).

In a study with barnacle cyprids, treatment of glass settling plates with DDT generally resulted in lowered settlement densities of the cyprids of *Balanus improvises* and lowered indices of preference for roughened surfaces. The results of this study suggest that DDT influences cyprid behavior by interfering with the rugophilic response pattern to settlement surfaces (Meith-Avcin, 1974).

Fiddler crabs (*Uca pugnax*) were fed natural detritus containing DDT residues (10 mg/kg) during an 11-day experiment. By day 5, the crabs were uncoordinated and sluggish in response to threat. DDT residues increased threefold during the experiment (Odum et al., 1969).

In other chronic toxicity tests with fiddler crabs (*Uca pugnax* and *U. pugilator*) effects on crab limb regeneration time were demonstrated at waterborne DDT concentrations of 10  $\mu\text{g/L}$  (Vernberg et al., 1977, as cited by Ballou et al., 1985).

## 6.2.2 DDE

In mammalian species, DDE is formed by the dehydrochlorination of the trichloroethane part of the DDT molecule (i.e., removal of hydrogen and chlorine from adjacent carbon atoms, resulting in a double bond) (BEIA, 1989). The p,p'-isomer of DDE is the most stable and persistent in tissues whereas the o,p'-isomer is more readily

eliminated. The half-life of elimination of DDE in pigeons is 250 days (Bailey et al., 1969a, b).

#### **6.2.2.1 Acute Effects**

DDE has an oral LD<sub>50</sub> of 880 mg/kg in male rats and 1,240 mg/kg in female rats (BEIA, 1989). Hill et al. (1975) performed sub-acute toxicity tests of DDE in several wildlife species to measure a median lethal dietary concentration (LC<sub>50</sub>). The 8-day test consisted of 5 days of treated diet followed by 3 days of untreated diet. The LC<sub>50</sub> in bobwhites was 825 mg/kg, the LC<sub>50</sub> in Japanese quail was 1,355 mg/kg, the LC<sub>50</sub> in ring-necked pheasant was 829 mg/kg, and the LC<sub>50</sub> in the mallard was 3,572 mg/kg.

EPA (1986) has stated that for saltwater test species, DDE concentrations as low as 14 µg/L have demonstrated acute toxicity in laboratory tests.

#### **6.2.2.2 Sub-Acute and Chronic Effects**

Like DDT, DDE also exhibits some estrogenic effects, with the o,p'-isomer being the most potent (BEIA, 1989). Tomatis et al. (1974) reported a reduced lifespan in both male and female mice treated with 250 µg/g p,p'-DDE in the diet for 130 weeks. Hepatic toxicity has also been reported in long-term DDE exposure to rats (BEIA, 1989).

Field and experimental evidence indicates that declines in eggshell thickness observed in certain species in North America and Great Britain since the mid-1940s have been largely caused by residues of p,p'-DDE or other compounds or metabolites of the DDT group (Cooke, 1973; Ohlendorf et al., 1978). At moderate or high levels of DDE, shell thinning is severe, and eggs may break during incubation. High DDE levels have been recorded in California; species affected have included brown pelicans (*Pelecanus occidentalis*) (Risebrough et al., 1971), double-crested cormorants (Gress et al., 1973),

great egrets (*Casmerodius albus*), and great blue herons (*Ardea herodias*) (Faber et al., 1972).

The thinning of eggshells of the brown pelican has proven to be related to the concentrations of DDE in the eggs (Blus et al., 1971; Blus et al., 1972a, 1972b). Nearly all brown pelican eggs collected from 13 colonies in South Carolina, Florida, and California in 1969 and from 17 colonies in South Carolina and Florida in 1970 exhibited eggshell thinning (Blus, 1970; Blus et al., 1974a). Of the 100 eggs analyzed for pollutant residues, all eggs contained measurable quantities of DDE; most eggs contained measurable quantities of DDD, DDT, dieldrin, or PCBs. DDE appears to have been responsible for virtually all the eggshell thinning.

Nest success of brown pelicans in South Carolina was related to residues of DDE and dieldrin in sample eggs (Blus et al., 1974b). DDE residues seemed primarily responsible for nest failure; however, deleterious effects of this pollutant on nest success were not separated satisfactorily from those induced by dieldrin. Significant intercorrelation of all five organochlorine residues identified in the eggs complicated the relationship of residues to nest success. Reproductive success in the brown pelican colony was subnormal in the 2 years of study (1971 and 1972) but reproductive success was normal in those nests in which the sample egg contained either 2.5 mg/kg or less of DDE, or 0.54 mg/kg or less of dieldrin.

Eggshell thinning has occurred in several other species that occur in freshwater or estuarine habitats or that nest on coastal islands. In 1967, shell thickness in herring gull (*Larus argentatus*) eggs from five states decreased with increases in chlorinated hydrocarbon residues (Hickey and Anderson, 1968). Comparison of eggshells taken before 1946 with those taken since then reveals that several species including the peregrine falcon (*Falco peregrinus*), brown pelican, double-crested cormorant, black-crowned night heron (*Nycticorax nycticorax*), bald eagle (*Haliaeetus leucocephalus*), and osprey (*Pandion haliaetus*) have sustained shell-thickness and shell-weight decreases of

20 percent or more, at least for brief periods (Anderson and Hickey, 1972). In some of these, regional population declines are known.

Shell thickness was significantly and inversely correlated with the concentration of DDE in 40 great blue heron eggs from Alberta (Vermeer and Reynolds, 1970; Vermeer and Risebrough, 1972).

In the Upper Great Lakes states, nine of 13 species of fish-eating birds were found in 1969-1970 to have sustained statistically significant decreases in eggshell thickness since 1946 (Faber and Hickey, 1973). Maximum changes in a thickness index occurred in great blue herons (-25 percent), red-breasted mergansers (*Mergus serrator*, -15 percent), and double-crested cormorants (-15 percent).

Studies with captive birds exposed to dietary DDE have shown the eggshell thinning and other reproductive effects observed in the wild. Longcore et al. (1971a and b) fed black ducks (*Anas rubripes*) diets containing DDE at 10 or 30 mg/kg (dry weight). The ducks experienced significant shell thinning, changes in mineral composition of eggshells, and an increase in shell cracking when compared with eggs of untreated black ducks. Survival of ducklings from dosed parents was 40 to 76 percent lower than survival of ducklings from undosed parents. Average DDE residues in eggs from hens fed 10 and 30 mg/kg DDE were 46 mg/kg and 144 mg/kg.

In another experiment, black duck hens fed 10 mg/kg (dry weight) of DDE in the diet laid eggs with shells more than 20 percent thinner than those of controls (Longcore and Samson, 1973). Natural incubation increased shell cracking more than fourfold as compared with mechanical incubation. Hens were observed removing cracked eggs from nests, and one hen terminated incubation. Hens fed DDE produced one-fifth as many ducklings as did the controls. The DDE in eggs of dosed hens averaged 64.9 mg/kg.

Like the black duck, the mallard is sensitive to DDE and DDT (Ohlendorf et al., 1978). Heath et al. (1969) observed significant eggshell thinning and cracking and a marked increase in embryo mortality in penned mallards fed 10 and 40 mg/kg DDE (dry weight). Other studies have been conducted by Longcore et al. (1971a), Tucker and Haegele (1970), Davison and Sell (1974), and Haegele and Hudson (1974). Those studies show effects on eggshell quality at dietary concentrations of 5 or 10 mg/kg of DDE.

Weimeyer et al. (1986) observed shell thinning in the American kestrel fed 10 mg/kg (dry weight) for one year. Eggshells were 10 percent thinner than controls, and reproductive success was strongly correlated to DDE dietary levels. McLane and Hall (1972) examined the effects of DDE on screech owl (*Otus asio*) reproduction. The owls received the dietary equivalent of 10 ppm DDE (dry weight) through one breeding season, which followed a year of no treatment. These birds laid eggs that were 12 percent thinner than eggs from the same birds fed untreated food a year earlier. Their eggs were also 13 percent thinner than untreated controls over the same 2-year period. In captive barn owls (*Tyto alba*) fed 2.83 mg/kg DDE (wet weight), alone or in combination with dieldrin (0.58 mg/kg, wet weight), DDE was associated with significant eggshell thinning, egg breakage, embryo mortality, and reduced production per pair (Mendenhall et al., 1983).

Wild-trapped starlings (*Sturnus vulgaris*) were fed concentrations of DDE or Aroclor 1254 (5, 25, and 100 mg/kg, dry weight) that were found to be sublethal when fed to penreared coturnix quail for 12 weeks (Dieter, 1975). After feeding for 7 weeks, the quails' liver residues of either organochlorine compound were about threefold higher than the concentrations fed daily. However, four times as much DDE as Aroclor 1254 had accumulated in the carcasses.

Greichus and Hannon (1973) studied the distribution and biochemical effects of DDT, DDD, and DDE in penned double-crested cormorants treated with 2, 5, and 10 mg of a combination of these compounds daily in their diet. Birds stressed by a one-half

decrease in food after the cessation of 9 weeks of treatment and birds that died of DDT toxicity showed a marked increase in brain and liver residues and a decrease in carcass residues. Liver and heart weights were significantly reduced because of treatment but not brain or spleen weights.

The effect of chronic sublethal dosages of DDE on the avoidance response of coturnix quail chicks was studied by Kreitzer and Heinz (1974). The chicks were on dosage beginning at 7 days of age for 8 days and on untreated feed for 6 days. Their avoidance response to a moving silhouette was measured daily for 14 days. No effect of DDE on the birds' behavior could be detected.

In toxicity studies with a dinoflagellate algal species (*Exuviella baltica*) DDE concentrations of 0.1  $\mu\text{g/L}$  and 10  $\mu\text{g/L}$  inhibited cell growth and inhibited cell production respectively (Powers et al., 1975).

### 6.2.3 DDD

In general, DDD is less toxic to animals than DDT. The effects of DDD poisonings are slower in onset, but longer in duration. In contrast to DDT, lethargy is more prominent and convulsions are less frequent (Grant, 1974). DDD is a primary metabolite of DDT and is further broken down to DDA, which is readily excreted in the urine either unchanged or as various metabolites (BEIA, 1989). The o,p'-isomer is most active on the liver, where it stimulates hepatic microsomal oxygenation of exogenous and endogenous compounds. DDD also has a unique ability to affect the adrenal gland. The half-life of elimination in pigeons is 24 days (Bailey et al., 1969a and b).

#### 6.2.3.1 Acute Effects

The oral  $\text{LD}_{50}$  in rats is reported as 113 mg/kg by RTECS (1989) and an oral  $\text{LC}_{50}$  value of 3,400 mg/kg was reported by Meister (1989). During 5-day dietary exposures,

the  $LC_{50}$  for birds ranged from 445 mg/kg in pheasants to 4,814 mg/kg in mallards (Hill et al., 1975)

EPA (1986) has stated that for saltwater test species, DDD concentrations as low as 3.6  $\mu\text{g/L}$  have demonstrated acute toxicity in laboratory tests.

#### **6.2.3.2 Sub-Acute and Chronic Effects**

DDD does not exhibit the same estrogenic effects that are exhibited by DDT (BEIA, 1989). Chronic feeding of DDD can result in liver, lung, and thyroid tumors in mice and rats (BEIA, 1989). However, chronic feeding studies have not been conducted for birds.

#### **6.2.4 BHC Isomers**

Lindane (i.e., gamma-BHC) is a neurotoxicant. By interfering with chloride transmission, lindane acts as a central nervous system stimulant. Small amounts of lindane may cause dizziness, nausea, muscle weakness, and tremors, while a massive dose results in vomiting and diarrhea, progressing to convulsions (BEIA, 1989). Circulatory and respiratory failure may also appear. The rate at which symptoms occur after ingestion of BHC isomers varies with isomer. Gamma-BHC evokes symptoms within 1 hour, alpha-BHC within 24 hours, and a commercial mixture within 2 to 12 hours after exposure (Gosselin, 1984). Young animals are more susceptible to the effects of lindane than adults (Clarke, 1981).

##### **6.2.4.1 Acute Effects**

In the rat, the oral  $LD_{50}$  is 76 mg/kg (BEIA, 1989). Hill et al. (1975) performed toxicity tests of lindane in several wildlife species to measure a median  $LC_{50}$ . The 8-day test consisted of 5 days of treated diet followed by 3 days of untreated diet. The  $LC_{50}$  in

the bobwhite (*Colinus virginianus*) was 882 mg/kg, the  $LC_{50}$  in the Japanese quail was 425 mg/kg, the  $LC_{50}$  in the ring-necked pheasant was 561 mg/kg, and the  $LC_{50}$  in the mallard was >5,000 mg/kg.

The acute oral  $LD_{50}$  of BHC in female mallards was  $\geq 1,414$  mg/kg and in pheasants it was 118 mg/kg; whereas the  $LD_{50}$  of lindane in male mallards was >2,000 mg/kg (Hudson et al., 1984). Mallards and pheasants exposed to these chemicals at ages 3 to 4 months showed a wide range of signs of neurotoxicity. Emaciation, enlarged livers, and small spleens were observed on necropsy of mortalities and sacrificed survivors.

Blakley (1982) studied the effects of lindane poisoning in a flock of Birmingham Rolles pigeons. Soon after ingestion of a commercial whole grain pigeon feed contaminated with 2,100 mg/kg lindane, the pigeons exhibited diarrhea, vomiting, anorexia, depression, and sudden death. This episode of acute lindane toxicosis caused sudden death in 47 percent of the pigeon flock.

Acute toxicity tests of technical and reference-standard lindane with saltwater crustaceans have indicated that  $LC_{50}$ s range from 0.17  $\mu\text{g/L}$  for the commercial pink shrimp (*Penaeus duorarum*) to 6.3  $\mu\text{g/L}$  for mysid shrimp (*Mysidopsis bahia*) (ASTER, 1992). Other reported acute  $LC_{50}$  values for saltwater invertebrates range from 450  $\mu\text{g/L}$  for American oyster (*Crassostrea virginica*) to 3,680  $\mu\text{g/L}$  for the annelid polychaete worm (*Neanthes arenaceodentata*). The  $LC_{50}$ s reported for lindane for saltwater fish range from 7.3  $\mu\text{g/L}$  for striped bass (*Morone saxatilis*) to 103.9  $\mu\text{g/L}$  for sheepshead minnow (*Cyprinodon variegatus*) (ASTER, 1992).

Laboratory acute toxicity tests with BHC (21 percent alpha, 39 percent gamma, 2.1 percent beta, 23 percent delta, and 14.9 percent unidentified) and pink shrimp and pinfish (*Lagodon rhomboides*) have resulted in acute  $LC_{50}$  values that were approximately 0.5 (0.34  $\mu\text{g/L}$ ) to 0.35 (86.4  $\mu\text{g/L}$ ) times less toxic than lindane, respectively.

#### 6.2.4.2 Sub-Acute and Chronic Effects

Wolfe and Esher (1980) studied the chronic toxicity of lindane in the old-field mouse (*Peromyscus polionotus*) and the cotton mouse (*P. gossypinus*). The feeding level was 0.2 mg lindane per gram of food for 8 months. Survival, reproduction, growth and development of young, and some aspects of behavior were not adversely affected by chronic exposure to lindane. The only significant result was the higher production of the number of litters by lindane-treated animals.

The effects of lindane on limb regeneration in the penaeid prawn was studied by Reddy and Rao (1989). Lindane inhibited limb regeneration in a dose-dependent manner. A concentration of 0.01 mg/L caused complete inhibition of regeneration and also delay in the initiation of limb bud regeneration.

In laboratory toxicity tests with lindane using saltwater phytoplankton communities, a 28.5 percent decrease in productivity as measured by C-14 were determined at 1,000  $\mu\text{g/L}$ . Other alga toxicity tests with *Acetabularia mediterranea* resulted in inhibition of cell growth and morphogenesis at a lindane concentration of 10,000  $\mu\text{g/L}$  (ASTER, 1992).

#### 6.2.5 Benzene

Benzene causes CNS depression, narcosis, and death in various species of animals (BEIA, 1989). Exposed animals may experience other CNS effects such as excitation, tremors, and loss of pupil reflexes. More subtle CNS effects include impaired learning ability and behavioral disturbances. In addition to its neurotoxic effects, benzene causes hematotoxicity and immunotoxicity. Animal laboratory studies suggest that benzene is carcinogenic.

### 6.2.5.1 Acute Effects

The most common routes of benzene exposure are inhalation and ingestion. An  $LC_{50}$  value of 10,000 ppm, 7-hour was recorded for the rat (TDB, 1984). Oral  $LD_{50}$  values of 4,700 mg/kg and 3,800 mg/kg have been reported for the mouse and rat, respectively.

Laboratory toxicity studies with benzene and saltwater organisms indicate that this compound is relatively less acutely toxic than most other contaminants of concern. Acutely lethal concentrations ranging between 17.6 to 924 mg/L have been determined for the invertebrates: Bay shrimp (*Crangon franciscorum*) and Pacific oyster (*Crassostrea gigas*), respectively.  $LC_{50}$  values for striped bass range from 5.1 to 10.9 mg/L depending on laboratory exposure methodology (ASTER, 1992).

### 6.2.5.2 Sub-Acute and Chronic Effects

The target cells for benzene-induced toxicity appear to be the cells of the bone marrow (BEIA, 1989). Leukopenia (i.e., reduction of white blood cells) is the most common manifestation of chronic benzene toxicity in laboratory animals. Leukopenia was observed in rats given 132 daily oral doses ranging from 10 to 100 mg/kg benzene (Wolf et al., 1956) and in mice administered benzene (400 and 600 mg/kg) in corn oil for 17 weeks (NTP, 1986). After inhalation of 17.5 ppm for 127 days, no blood changes were observed in rats, guinea pigs, and dogs. Slight leukopenia has been reported in rats exposed to 44 ppm, 5 hours per day, 4 days per week for 5 to 7 weeks (IARC, 1983).

Benzene has been widely tested in a number of laboratory animals to assess potential reproductive effects. When administered orally to pregnant mice at doses at 0.5 and 1.0 mg/kg body weight, benzene did not induce fetal malformations but did cause maternal lethality and resorptions (Nawrot and Staples, 1979). At the dose of 1.47 ml/kg/day, benzene caused significant reductions in fetal body weights (Seidenberg

et al., 1986). Teratogenic effects were not observed in rats, rabbits, or mice when tested by inhalation, even at dose levels that were toxic to the mother (125 to 940 ppm) as evidenced by her reduced weight gain (ATSDR, 1987). However, benzene at concentrations of 100 to 940 ppm was fetotoxic, causing resorptions, reduced fetal weights, and skeletal variations.

Levels of benzene required to induce chronic, sublethal responses in saltwater organisms have been determined at much lower concentrations than acute exposures. In toxicity tests with blue crabs, 400  $\mu\text{g/L}$  benzene increased the time to molt in juvenile crabs (Cantelmo et al., 1982, as cited by Ballou et al., 1985). Sublethal effects of benzene on physiological and developmental functions in market crab (*Cancer magister*), pacific oyster, and mussels (*Mytilus edulis*) have been reported at concentrations ranging from 1,100 to 5,000  $\mu\text{g/L}$ .

Other chronic exposure effects of benzene on saltwater algal species have been determined at much higher concentrations. Growth potential in marine dinoflagellate and diatoms were inhibited at concentrations of benzene ranging from greater than 5,000 to 100,000  $\mu\text{g/L}$  (ASTER, 1992).

In chronic sublethal toxicity tests with saltwater fish, benzene concentrations of 113  $\mu\text{g/L}$  induced changes in hematological (blood) parameters in striped bass after a 4-hour exposure (ASTER, 1992). In a 28-day exposure to benzene at 3,100  $\mu\text{g/L}$ , striped bass had significant changes in physiological processes. In other longer-term toxicity tests with Pacific herring (*Clupea harengus pallasii*), stress on the test fish was noted after 6 days, and reduced survival was seen after 7 days at 700  $\mu\text{g/L}$  of benzene (ASTER, 1992).

## 6.2.6 Chlorobenzene

The toxic effects of chlorobenzene are similar to those of the chlorinated hydrocarbons. In particular, the chemical is a CNS depressant (Deichmann, 1981). Symptoms of toxicity include transient ataxia, labored breathing, prostration, and hyperpnea. Chlorobenzene can also cause narcosis (BEIA, 1989). There is some evidence for carcinogenic activity of chlorobenzene in high-dose male rats, but the results of this study are not definitive (EPA, 1988).

### 6.2.6.1 Acute Effects

Single oral doses of chlorobenzene were lethal at levels of 4,000 mg/kg in male and female rats,  $\geq 1,000$  mg/kg in male mice, and  $\geq 2,000$  mg/kg in female mice (EPA, 1988). Most deaths occurred within a few days of administration.

The acute toxicity of chlorobenzene to mysid shrimp (*Mysidopsis bahia*) and sheepshead minnows have been determined to be 16.4 and 10.5 mg/L, respectively (ASTER, 1992). In acute tests with the saltwater diatom (*Skeletonema costatum*) the 96-hour  $EC_{50}$  (effects concentration) for Chlorophyll A and cell numbers was determined to be 343 and 341 mg/L, respectively.

### 6.2.6.2 Sub-Acute and Chronic Effects

Chronic administration of chlorobenzene produces pathological changes of the liver and kidneys (BEIA, 1989). Dose-dependent necrosis of the liver; degeneration or focal necrosis of the renal proximal tubules; and lymphoid depletion of the spleen, bone marrow, and thymus were produced by chlorobenzene at oral doses of 250 mg/kg or greater in both sexes of rats and mice (Kluwe et al., 1985). Toxic effects were not observed at 125 mg/kg or less.

John et al. (1984) evaluated the teratogenic, embryotoxic, and reproductive effects of chlorobenzene in rats and rabbits. Vapor concentrations were 75, 210, and 590 ppm (345, 966, or 2,714 mg/m<sup>3</sup>) 6 hours per day on gestational days 6 through 15 for rats and 6 through 18 for rabbits. In rats and rabbits, maternal liver weights were elevated, and body weights and feed consumption were decreased in maternal rats, but teratogenic and embryotoxic effects were not observed in the offspring of rats or rabbits. In a two-generation study by Nair et al. (1987), male and female rats were exposed by inhalation to vapors of chlorobenzene at concentrations of 50, 150, or 450 ppm. No adverse effects on reproductive performance or fertility of the males or females were observed; however, dose-dependent bilateral degeneration of the testicular germinal epithelium was observed in adult males and male offspring. In addition, dose-dependent hepato-cellular and renal changes were in the same groups of rats.

An inhalation study with dogs indicated bilateral atrophy of the epithelial tissue in the seminiferous tubules in two of four dogs exposed to 2 mg/L chlorobenzene vapor for 6 hours/day, 5 days a week for a total of 62 exposures. This effect was not seen in similarly exposed dogs at the concentration of 1.47 mg/L (Monsanto, 1978).

Although no chlorobenzene chronic toxicity studies with saltwater test species were found, chronic values for 1,2,4-trichlorobenzene and 1,2,4,5-tetrachlorobenzene were determined to be 222 and 129 µg/L, respectively, for sheepshead minnows (ASTER, 1992).

## **6.2.7 Chloroform**

Chloroform is a CNS depressant that is toxic to the liver and kidneys, and carcinogenic in laboratory animals (BEIA, 1989).

#### 6.2.7.1 Acute Effects

The acute toxicity of chloroform in laboratory animals depends upon species, strain, and sex. Oral LD<sub>50</sub> values ranged from 36 to 1,400 mg/kg in the mouse to 900 to 2,000 mg/kg in the rat (RTECS, 1984; Perwak et al., 1980). Oral LD<sub>50</sub> values in male mice varied from 120 mg/kg in DBA/2J mice to 490 mg/kg in C57BL/6J mice. LC<sub>50</sub> values of 8,000 ppm • 4 hours, 20,000 ppm • 2 hours, and 35,000 ppm • 4 hours have been recorded for the rat, guinea pig, and cat, respectively (RTECS, 1984).

The 96-hour acute LC<sub>50</sub> chloroform concentration for pink shrimp (*Penaeus duorarum*) has been determined to be 81.5 mg/L (EPA, 1980d).

#### 6.2.7.2 Sub-Acute and Chronic Effects

The primary target organs of chronic chloroform exposure are the kidney and liver. In a study by Torkelson et al. (1976), several species were exposed to chloroform vapors 7 hours per day, 5 days per week for 6 months. Toxic effects, which were dose- and species-dependent, included increased kidney weight, degeneration of liver, decreased body weight, and changes in the lung.

Reproductive studies of chloroform have given mixed results. Schwetz et al. (1974) observed dose-dependent reproductive toxicity in rats exposed to chloroform vapors of 30,100 or 300 ppm, 7 hours daily on days 6 to 15 of gestation. At 100 ppm, chloroform caused a significant incidence of tail absence or shortening, imperforate anus, subcutaneous edema, missing ribs, and delayed ossification of sternebrae. At 300 ppm, a decrease in conception rate and a high incidence of fetal resorption were observed. Other studies, however, have obtained negative reproductive results in inhalation or gavage studies in rats, rabbits, and mice (BEIA, 1989).

## 6.2.8 1,2-Dichloroethane

High vapor concentrations of 1,2-DCA can produce irritation of the eyes, nose, and throat (BEIA, 1989). Ingestion or inhalation of the compound can cause CNS depression and systemic injury to the liver, kidneys, and lungs. 1,2-DCA is also carcinogenic in laboratory animals.

### 6.2.8.1 Acute Effects

Oral LD<sub>50</sub> values of 670, 860, and 5,700 mg/kg have been reported for rats, rabbits, and dogs, respectively (NIOSH, 1989). Spencer et al. (1951) found that rats survived a 12-minute exposure to 12,000 ppm, a 1-hour exposure to 3,000 ppm, and a 7-hour exposure to 300 ppm. Vapors of 1,2-DCA have been shown to cause reversible clouding of the corneas of dogs and foxes but not of other species (Grant, 1974).

The 96-hour acute LC<sub>50</sub> for 1,2-DCA for mysid shrimp (*Mysidopsis bahia*) is 113 mg/L (ASTER, 1992) for the sheepshead minnow. The 96-hour LC<sub>50</sub> is between 126 and 226 mg/L (ASTER, 1992). The 24- and 96-hour acute LC<sub>50</sub> concentrations of 1,2-DCA for sand shrimp (*Crangon crangon*) were determined to be 75 and 65 mg/L, respectively (Verschueren, 1983). An LC<sub>50</sub> of 185 mg/L for an exposure of 1 hour was determined for the goby (*Gobius minutus*), a saltwater fish.

Acute 96-hour EC<sub>50</sub> concentrations for the saltwater alga (*Skeletonema costatum*) were found to exceed 433 mg/L for both Chlorophyll A and cell numbers (ASTER, 1992).

### 6.2.8.2 Sub-Acute and Chronic Effects

Chronic exposure to chloroform vapor can result in systemic toxicity and death. Heppel et al. (1946) exposed various animals 7 hours per day, 5 days a week to 1,2-DCA vapor concentrations ranging from 100 to 1,000 ppm. Pathological

examination showed pulmonary congestion, renal tubular degeneration, fatty degeneration of the liver, and, less commonly, necrosis and hemorrhage of the adrenal cortex and fatty infiltration of the myocardium.

Although placental transfer of 1,2-DCA appears to occur (BEIA, 1989), reproductive studies have given mixed results concerning the embryotoxic, fetotoxic, teratogenic, and maternal toxicity of 1,2-DCA. Litter size, birth weight, and peri- and postnatal survival were reduced significantly in rats exposed to 1,2-DCA vapor at a concentration of 14 ppm for 4 hours per day for 6 months (Vozovaya, 1974). Maternal fertility was decreased, and the estrous cycle was lengthened. No embryotoxicity, fetotoxicity, or malformations, however, were observed in rats exposed to 100 and 300 ppm, 1,2-DCA for 7 hours daily on days 6 through 15 of gestation even though severe maternal toxicity was observed in high-dose rats (Rao et al., 1980). At 25, 75, and 150 ppm, no adverse effects on the reproductive capacity of the adult rats or on growth and survival of the offspring were noted.

When administered via the drinking water or feed, 1,2-DCA does not appear to produce dose-dependent effects on fertility, gestation, litter size, fetal weight, fetal development, or viability of offspring (BEIA, 1989).

## **6.2.9 Ethylbenzene**

Ethylbenzene is primarily an irritant to the skin, eyes, and upper respiratory tract (BEIA, 1989). Systemic effects include CNS depression and edema and hemorrhage of the lung.

### **6.2.9.1 Acute Effects**

The oral LD<sub>50</sub> of ethylbenzene in rats is 3,500 mg/kg; dermal LD<sub>50</sub> values of 500 mg/kg and 17,800 mg/kg have been reported (BEIA, 1989).

The acute  $LC_{50}$  for ethylbenzene in saltwater shrimps (*Mysidopsis bahia* and *Cragon franciscanum*) are 87.6 and 3.7 mg/L, respectively. The 24-hour  $LC_{50}$ s for grass shrimp (*Palaemonetes pugio*) adults and larvae were determined to be 14.4 and 10.2 mg/L, respectively (ASTER, 1992). The 96-hour  $LC_{50}$  for the market crab was found to be 13.0 mg/L (Caldwell and Mallon, 1977, as cited in Ballou et al., 1985). In other 96-hour acute toxicity tests with bay shrimp an  $LC_{50}$  value of 0.49 was determined (EPA, 1980f). The Pacific oyster had a much greater  $LC_{50}$  at 1,030 mg/L (ASTER, 1992).

Two saltwater fish species have been tested in the laboratory, resulting in 96-hour  $LC_{50}$  values of 275 mg/L (sheepshead minnow) and 430 mg/L (striped bass) (EPA, 1980f). In other acute toxicity tests with coho salmon, 50 mg/L and 10 mg/L ethylbenzene resulted in 100 percent and 6.7 percent mortality, respectively, after 24 hours of exposure.

The 96-hour acute  $EC_{50}$ s for the alga (*Skeletoema costatum*) for Chlorophyll A and cell number was found to be greater than 438 mg/L ethylbenzene (ASTER 1992).

#### **6.2.9.2 Sub-Acute and Chronic Effects**

Chronic inhalation exposure of guinea pigs, monkeys, rabbits, and rats at concentrations ranging from 400 to 2,200 ppm for 7 to 8 hours per day, 5 days per week for 6 months produced no effects in any of the animals tested except for a slight increase in liver and kidney weights of rats (Wolf et al., 1956). No effects on the bone marrow were observed.

Maternal toxicity in rats but not rabbits was observed by Hardin et al. (1981) at an exposure level of 1,000 ppm ethylbenzene for 6 to 7 hours daily. Ungvary and Tatrai (1985) observed dose-dependent embryo toxicity in rats, mice, and rabbits. Some effects included maternal toxicity, an increase in post-implantation loss, and skeletal retardation in rats (600, 1,200, and 2,400 mg/m<sup>3</sup>); uropoietic anomalies in mice

(500 mg/m<sup>3</sup>); and abortion, fetal resorption, and maternal death in rabbits (1,000 mg/m<sup>3</sup>).

Sublethal effects of excess mucous production in the manila clam (*Tapes semidecussata*) were determined at an ethylbenzene concentration of 0.08 µg/L (Nunes and Benville, 1979, as cited by Ballou et al., 1985).

## **6.2.10 Toluene**

Inhalation appears to be the most frequent and most important route of exposure to toluene (BEIA, 1989). The main toxic effects are upon the CNS. At high levels of exposure, toluene can cause narcosis and death.

### **6.2.10.1 Acute Toxicity**

The minimum lethal vapor concentration for mice was found to be 5,300 ppm in an 8-hour exposure (NIOSH, 1973). The inhalation LC<sub>50</sub> value for mice is 5,320 ppm • 8-hour (RTECS, 1984). Acute oral and dermal exposure are also possible. The oral LD<sub>50</sub> for rats is 5,000 mg/kg (RTECS, 1984). Dermal LD<sub>50</sub> values of 12,100 and 14,000 mg/kg have been reported for the rabbit (Clayton, 1981; RTECS, 1984).

The reported acute toxicity values of toluene to saltwater crustaceans include 96-hour LC<sub>50</sub>s of 3.7 mg/L (bay shrimp), 9.5 mg/L (grass shrimp larvae, *Palaemonetes pugio*), 28 mg/L (Stage I—market crab larvae), and 56.3 mg/L (mysid shrimp, *Mysidopsis bahia*) (ASTER, 1992). Other acute toxicity studies with crustaceans have determined 24-hour LC<sub>50</sub> values for the saltwater copepod (*Nitocra spinipes*) to range between 24.2 and 74.2 mg/L (ASTER, 1992).

The Pacific oyster is reported to have an acute LC<sub>50</sub> concentration of 1,050 mg/L for toluene (ASTER, 1992). For saltwater fish species acute LC<sub>50</sub> concentrations of

6.3 mg/L for striped bass and greater than 277 and less than 485 mg/L for sheepshead minnows have been reported (ASTER, 1992). Other fish acute toxicity results include an  $LC_{50}$  of 5.5 mg/L for coho salmon (EPA, 1992g). However, other researchers found no mortality to coho salmon at toluene concentrations up to 10 mg/L for 96 hours (Morrow, 1974, as cited by Verschueren, 1983).

#### **6.2.10.2 Sub-Acute and Chronic Effects**

After chronic oral exposure to toluene, species- and dose-dependent effects may be observed on the liver, kidney, heart (increased weight), brain (necrosis), and urinary bladder (hemorrhage) (BEIA, 1989). Similar effects are observed after inhalation. Studies in which toluene has been administered by inhalation to pregnant test animals have shown that toluene can elicit teratogenic and embryotoxic effects. Ungvary (1985) noted signs of skeletal retardation in offspring of pregnant rats exposed to 1,000 mg/m<sup>3</sup>. Courtney et al. (1986) administered 1,500 mg/m<sup>3</sup> to mice from days 6 to 16 of gestation and considered it teratogenic at that level because of a significant shift in the fetal rib profile.

Following oral exposure of toluene, doses of 0.3, 0.5, or 1.0 ml/kg, embryonic lethality was observed, and fetal weights were reduced at the two higher doses (Nawrot and Staples, 1980). A significant increase in the incidence of cleft palate occurred at the 1.0 ml/kg level.

In chronic sublethal toxicity tests with Manila clam, Pacific oyster, and California mussel (*Mytilis californianus*) effect concentrations of 1.3, 3.1, and 100.0 mg/L were determined for those species, respectively (Legone, 1974; Nunes and Benville, 1979; and Sabrurin and Tullis, 1981, all as cited in Ballou et al., 1985).

Additionally, in studies with coho salmon after 1 hour of exposure to 1.65 mg/L of toluene, young fry demonstrated avoidance to the toxicant and at 40 days demonstrated reduced growth at 1.41 mg/L toluene (ASTER, 1992).

Algal test species have reported  $EC_{50}$ s or demonstrated effects at from 8.0 mg/L (for reduction of growth potential in the alga (*Skeletonema costatum*) to greater than 433 mg/L (for reduction in Chlorophyll A in the same species) (ASTER, 1992). Other algal tests have reported nonlethal effects ranging from 10 mg/L for the reduction of photosynthesis in the kelp (*Macrocystis pyrifera*) to 100 mg/L for growth potential inhibition for the algal species: *Cricosphaera carterae*, *Dunaliella tertiolecta*, and *Amphidinium carteri* (ASTER, 1992).

### 6.2.11 Xylene

Xylene exposure produces a narcotic effect on the CNS and has variable effects on the liver and kidneys (BEIA, 1989). It can also cause irritation of the gastrointestinal tract. Xylene does not cause myelotoxic effects.

#### 6.2.11.1 Acute Effects

An  $LC_{50}$  value of 500 ppm • 4-hour and oral  $LD_{50}$  value of 4,300 mg/kg have been reported for rats (RTECS, 1984). An  $LC_{50}$  value in cortunix quail has been reported to be >20,000 mg/kg (Hill and Camardese, 1986).

Acute 96-hour  $LC_{50}$  o-xylene concentrations of 7.4 mg/L, and 12.0 mg/L have been determined for grass shrimp larvae and market crab larvae, respectively (Caldwell and Mallon, 1977, and Tatum et al., 1978, as cited in Ballou et al., 1985). The m-xylene 96-hour acute  $LC_{50}$  for market crab was determined to be 12.0 mg/L (Verschueren, 1983). Bay shrimp 96-hour acute  $LC_{50}$  values for o-xylene, m-xylene, and p-xylene were determined to be 1.3, 3.7, and 2.0 mg/L, respectively (Verschueren, 1983).

For fish species, the striped bass 96-hour acute  $LC_{50}$  values for o-xylene, m-xylene, and p-xylene were found to be 11.0 mg/L, 9.2 mg/L, and 2.0 mg/L, respectively (Verschuere, 1983). In acute toxicity studies with young coho salmon, concentrations of up to 10 mg/L of o-xylene produced no mortality after 96 hours of exposure.

#### **6.2.11.2 Sub-Acute and Chronic Effects**

Animal laboratory studies indicate that xylene has a relatively low toxicity over the long term (BEIA, 1989). Some effects on the central nervous system may occur (such as CNS depression), and chronic xylene may elicit behavioral changes.

Ungvary et al. (1980) assessed the teratogenic activity of the xylene isomers. Rats were exposed by inhalation to 35, 350, or 700 ppm continuously on days 7 through 14 of gestation. Teratogenic effects were not observed. Maternal food intake was reduced at the highest exposure level, which resulted in decreased fetal development. In a later experiment by Ungvary and Tatrai (1985), in which rats, mice, and rabbits were exposed to xylene mixtures by inhalation, skeletal and weight retardation of fetuses was significant in mice at  $1,000 \text{ mg/m}^3$ . The same concentration was toxic to pregnant rabbits, causing death, abortion, or total resorption. In rats, skeletal retardation was found at  $250 \text{ mg/m}^3$ , and dead or resorbed embryos were found at  $3,400 \text{ mg/m}^3$ .

Following oral xylene exposure to mice, Marks et al. (1982) found no effects in either the dams or the fetuses at 520 or  $1,030 \text{ mg/kg/day}$ . However, doses of 2,060, 2,580, 3,100, and  $4,130 \text{ mg/kg/day}$  decreased fetal weights significantly and increased fetal malformations; cleft palates was the major malformation noted. Nawrot and Staples (1980) also noted a dose-dependent increase in cleft palates in mice exposed orally to xylenes.

The lowest concentration where sublethal development effects were demonstrated to Pacific oysters were determined to be 3.1 mg/L (xylenes), 1.22 mg/L (m-xylene), and

0.359 mg/L (for both o-xylene and p-xylene) (Legone, 1974, as cited by Ballou et al., 1985). Additionally, increased mucous production in Manila clams was found at an o-xylene concentration of 0.13 mg/L (Nunes and Benville, 1979, as cited by Ballou et al., 1985).

### 6.3 Bioaccumulation Potential

The term bioaccumulation can be defined as the uptake and accumulation of chemicals by organisms from the nonliving (abiotic) environment (e.g., water, sediment, soil, or air) or through the diet. Many compounds enter plant and animal tissues more readily than they leave, in part because once in cells they bond to or with organic compounds. This causes the concentration of the compound to be higher within the organism than outside it. The bioaccumulation of a chemical directly from the nonliving environment, which results in a greater whole-body concentration than that found in the environment, is known as bioconcentration. When bioaccumulation of chemicals occurs at ever greater concentrations from one trophic level to another through the food chain, it is called biomagnification.

A standard technique for reporting the potential for a chemical to bioconcentrate within an aquatic organism is the bioconcentration factor (BCF). The steady-state BCF is defined as the ratio of the concentration of a chemical in the tissues of an aquatic organism to the concentration of that chemical in water. This factor then provides a relative measure of the potential for a specific chemical to accumulate within an organism and its hazard to that and other exposed organisms.

Of the identified chemicals of concern, DDT, DDE, and DDD have the highest potential to bioaccumulate (Table 5-1, Appendix A). These compounds are nearly insoluble in water, undergo limited breakdown in the natural environment, and are highly lipophilic. DDT and its metabolites undergo all three processes of bioaccumulation,

bioconcentration, and biomagnification. Because lindane and BHC are more water soluble and less stable in the environment, the potential for these compounds to bioaccumulate is less than that of DDT and its metabolites. On the other hand, benzene, chlorobenzene, chloroform, 1,2-dichloroethane, ethylbenzene, toluene, and the xylenes have little or no tendency to bioaccumulate.

### 6.3.1 DDT, DDE, and DDD

DDT and its metabolites have a very high potential to bioconcentrate in aquatic plants and animals (Appendix A). For example, the BCF of DDT in mussels can reach values as high as 690,000 (Reish et al., 1978). The BCF of DDE in mosquito larvae is 59,000 (Callahan, 1979), and the BCF of DDD in oysters is 47,900 (Zarogian et al., 1985). The BCF of DDT in curly leaf pondweed is 14,280 (Eberhardt et al., 1971) while the BCF of DDE and DDD in algae is 2,720 and 6,210, respectively (Verschuere, 1983).

Earthworms can accumulate persistent soilborne insecticides and are an important source of contamination of terrestrial wildlife (Beyer and Gish, 1980; Beyer and Krynitsky, 1989). DDT was applied once to an experimental plot at a rate of 9 kg/ha, and concentrations were measured in earthworms over a 20-year period. DDE, the metabolite of DDT most significant to wildlife, increased until the third year and then decreased with a half-time of 5.7 years. The declining parts of the curve fit experimental decay equations reasonably well. This estimate of persistence was considered relevant to assessment of DDT at low or moderate concentrations in relatively undisturbed soils in temperate climates.

DDE has a high bioaccumulation potential in avian species. In a study by Dieter (1974), coturnix quail were fed 5, 25, and 100 mg/kg DDE dry weight in their diet for 12 weeks. DDE accumulated up to fourfold higher in the carcasses and livers than those in the diets. After feeding at the same dietary levels for 7 weeks, DDE liver residues in starlings were threefold higher than the concentrations fed daily (Dieter,

1975). Porter and Wiemeyer (1972) studied DDE in the diet of the American kestrel. Fourteen birds were fed 2.8 mg/kg dietary DDE for several months. In two birds that died after 14 and 16 months of feeding, the brain DDE residues were 301 and 218 mg/kg.

### 6.3.2 BHC and Lindane

Lindane is much less readily bioaccumulated than DDT by birds (Stickel, 1973). Because lindane was found to be safer than alternative organochlorine chemicals used as a seed dressing, it was proposed as an alternative chemical (Burrage and Saha, 1972). When such a shift was made (from heptachlor to lindane) in the Columbia Basin of Oregon and Washington, reproductive success of geese increased, mortality decreased, and the nesting population increased (Blus et al., 1984). There was no evidence for either biomagnification of lindane residues from treated seed to goose tissues or eggs or for induction of adverse effects by this compound.

Compared to DDT and its metabolites, lindane and BHC have a moderate potential to bioconcentrate in aquatic plants and animals (Appendix A). The BCF of lindane in edible tissue and offal tissue of the pinfish (*Lagodon rhomboides*) is 130 and 617, respectively, while the BCF for BHC is 490 in the sheepshead minnow. The BCF of lindane in the American oyster (*Crassostrea virginica*) is 218 (Shimmel et al., 1977); in grass shrimp (*Palaemonetes pugio*) and pink shrimp (*Penaeus duorarum*), the BCF of BHC is 63 and 84, respectively.

## 6.4 Known Effects in the Study Area

Limited ecological effects data are available for the Montrose drainage area. Information has been gathered from both routine monitoring and specific testing programs. The area has been recovering over a number of years from the heavy impacts

associated with the Dominguez Channel and harbor discharges. Disease (various carcinomas and abnormalities) and parasitism in fish from upper Los Angeles Harbor have been associated with these discharges. However, the incidence of these problems in Los Angeles Harbor fish, in general, has been decreasing since the 1970s (MEC, 1988). It is difficult to assign toxic effects observed in fish or benthic invertebrates in the Dominguez Channel or Consolidated Slip to specific Montrose-associated chemicals because the area has been so heavily affected by a variety of discharges. In addition, fish move readily among sites. In comparison, bioaccumulation information on sedentary invertebrates can assign specific types of contamination to specific areas.

Outside the study area, effects of DDT and its metabolites (especially DDE) have been documented in migratory birds along the Southern California Coast (EPA, 1975; Ohlendorf et al., 1978). These and other effects for areas outside the study area are being addressed by NOAA (NOAA, 1990 and 1991).

#### 6.4.1 Toxicity Tests

Limited sediment toxicity information exists for the Consolidated Slip as part of the dredging evaluation conducted during 1982 (Marine Bioassay Laboratories, 1982). By their nature, the toxicity tests do not specifically point to or exclude Montrose chemicals of concern as elements of toxicity. The toxicity of various sediment fractions from the Consolidated Slip is shown in Table 6-5. The sediment was toxic in liquid, suspended particulate, and solid phase tests. The solid and suspended sediment phase tests are particularly applicable for comparison to Montrose chemicals of concern. Mysid shrimp, fish, and sea urchins demonstrated toxicity in suspended phase tests, while mysids and worms showed solid phase toxicity (although bivalves did not). Bioaccumulation was tested during the same test series. The bivalve *Macoma nasuta* showed some accumulation of metals during a 10-day exposure to the sediments, but no accumulation of total chlorinated hydrocarbons or PCBs (Marine Bioassay Laboratories, 1982).

Table 6-5 Consolidated Slip Dredging Project Bioassay Results			
Test Organism	Sediment		
	Liquid Phase	Suspended Particulate Phase	Solid Phase
<i>Acanthomysis sculpta</i> (mysid)	T	T	T
<i>Citharichthys stigmaeus</i> (fish)	T	T	—
<i>Strongylocentrotus purpuratus</i> (sea urchin)	T	T	—
<i>Macoma nasuta</i> (clam)	—	—	NS
<i>Neanthes arenaceodentata</i> (worm)	—	—	T
Notes: NS = Non-significant result T = Demonstrated toxicity during test — = Not tested  Source: Clark, 1982			

#### 6.4.2 Benthic Community Structure

The preliminary benthic community sampling of the Dominguez Channel and Consolidated Slip provide some information on the condition of the natural communities. Seven to 11 benthic species were collected in the Consolidated Slip during 1978 quarterly samples. This compared to a list of 28 or 29 species for another Inner Los Angeles Harbor station and 37 to 43 species for Outer Los Angeles Harbor, collected as part of the same study (Clark, 1982). The Shannon-Wiener species diversity index ( $H'$ ), also was comparatively low for the Consolidated Slip. The Consolidated Slip  $H'$  ranged from 0.12 to 1.15, the Inner Harbor ranged from 1.46 to 1.86, and the Outer Harbor ranged from 2.02 to 2.16. Species collected on settling racks displayed the same general pattern of abundance and diversity, with the Consolidated Slip showing the greatest evidence of pollution effects (Clark, 1982). These results, taken in total, indicate that the benthic community of the Consolidated Slip is stressed by pollution as compared to comparable local sites. The type of pollutant stress cannot be determined from the community structure information.

Comparable diversity studies have not been conducted for the Dominguez Channel community, although the benthic invertebrate species list (Table 4-1) is similar to that of the Consolidated Slip (Table 4-4), indicating a similar, moderately pollutant-stressed benthic community.

### **6.4.3 Bioaccumulation**

California State Mussel Watch data provide some information on contaminant bioaccumulation in bivalves transplanted to the Montrose drainage area. Data compiled since 1982 show contaminant levels for DDT and BHC and associated compounds in transplanted California mussels at the Consolidated Slip and other sites further down into the Los Angeles Harbor ship channel and the main portion of Los Angeles and Long Beach Harbors. An additional site was assayed in the Dominguez Channel as part of a separate study (Young and Heeson, 1974). Recent average mussel tissue values are given in Table 6-6 on a dry weight, wet weight, and lipid weight standardized basis. There has been an area-wide trend toward decreasing total DDT bioaccumulation in mussels over time, including mussels at the Consolidated Slip (Figure 6-1). Similar decreasing concentrations of DDT in mussels have been reported for the Los Angeles River, Long Beach, and Royal Palms areas (Mearns et al., 1991). The decreasing total DDT values shown on Figure 6-1 may help explain the relatively high total DDT observed in the Dominguez Channel mussels in 1974 (Table 6-6). BHC data are limited and do not show as obvious a decrease in bioaccumulation as for DDT, although recent data are lower than those in the early 1980s (Figure 6-2). Other compounds from the list of Montrose chemicals of concern were not analyzed as part of the State Mussel Watch Program.

The potential for bioaccumulation and toxicity of many highly water-insoluble compounds (such as DDT and its metabolites) is greatly affected by the levels of organic carbon found in sediments where these chemicals are present (Pavlou, 1987; EPA, 1988). This is because the chemicals are associated with the organic matrix of

**Table 6-6**  
**Mussel Bioaccumulation of DDT and BHC Compounds**  
**in the Dominguez Channel and Consolidated Slip**

Year	Station	Compound	Concentration (µg/kg)		
			Dry Weight	Wet Weight	Lipid Weight
1974 <sup>a</sup>	Dominguez Channel	Total DDT	—	260	—
1987-1990 <sup>b</sup>	Consolidated Slip averages	Total DDT	479	19	10,829
		o,p'-DDD	22	2.4	566
		p,p'-DDD	113	15.4	2,720
		o,p'-DDE	38	4.9	852
		p,p'-DDE	247	33	5,339
		p,p'-DDMS	36 <sup>c</sup>	4.5 <sup>c</sup>	551 <sup>c</sup>
		p,p'-DDMU	26	2.3	641
		o,p'-DDT	ND	ND	ND
		p,p'-DDT	21	1.3	527
		alpha-BHC	1.2	—	30 <sup>d</sup>
		beta-BHC	ND	—	ND
		delta-BHC	ND	—	ND
		gamma-BHC	2.8 <sup>d</sup>	—	71 <sup>d</sup>

<sup>a</sup>From Young and Heeson, 1974.

<sup>b</sup>California Mussel Watch Data, Preliminary Reports, 1987-88, 1988-89, 1989-90.

<sup>c</sup>1988 value only.

<sup>d</sup>1987-88 value only.

ND = Nondetectable.

the sediment particles; they are therefore less likely to be present in the water column and are less bioavailable from ingested sediments. For these reasons, an equilibrium partitioning approach to bioaccumulation potential that takes the organic carbon content of sediments into account appears to be the preferred method for assessing potential effects of contaminants of concern in the study area. However, very little data for organic carbon content is available for sediments in the study area (see Section 7.1.2).

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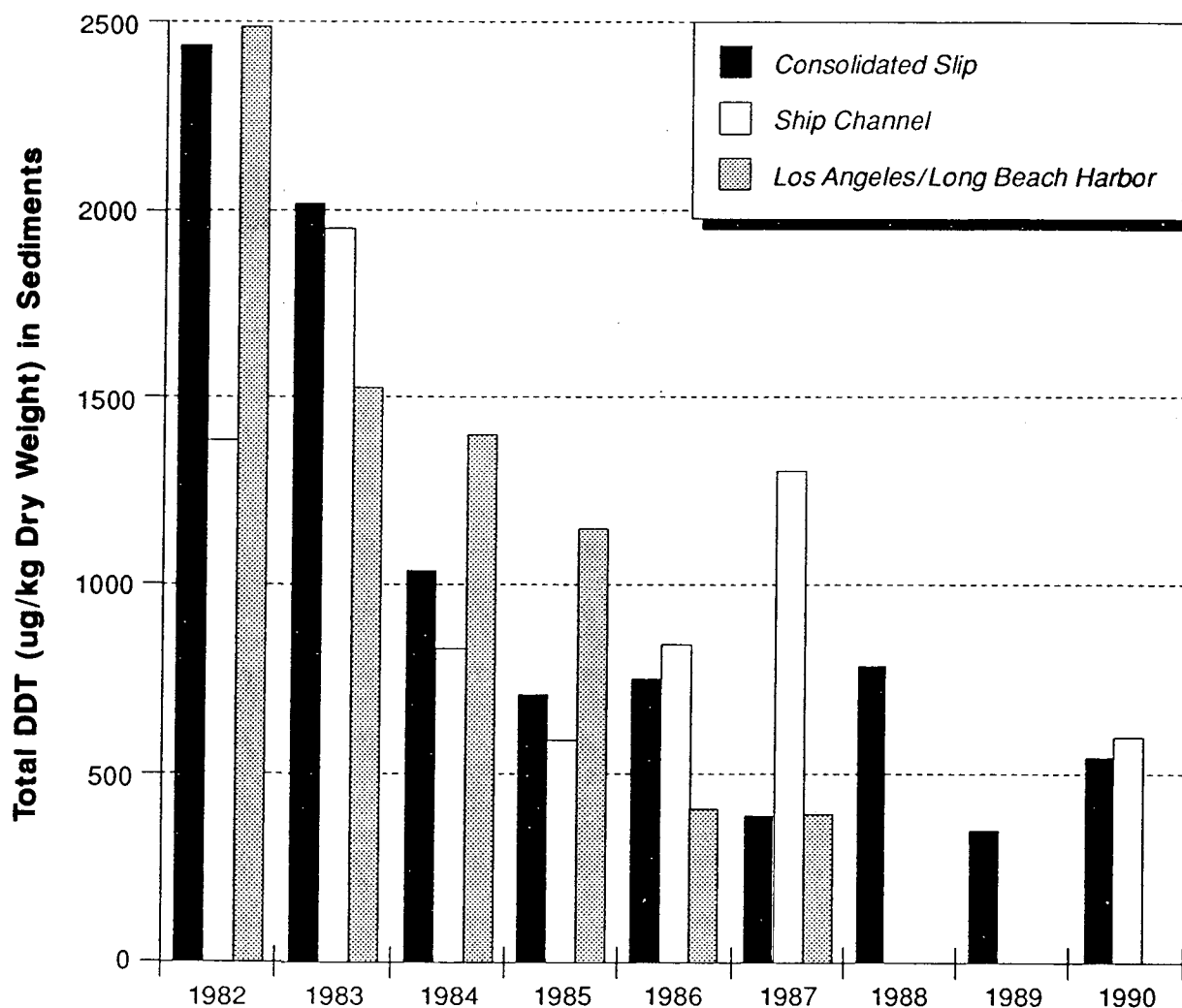
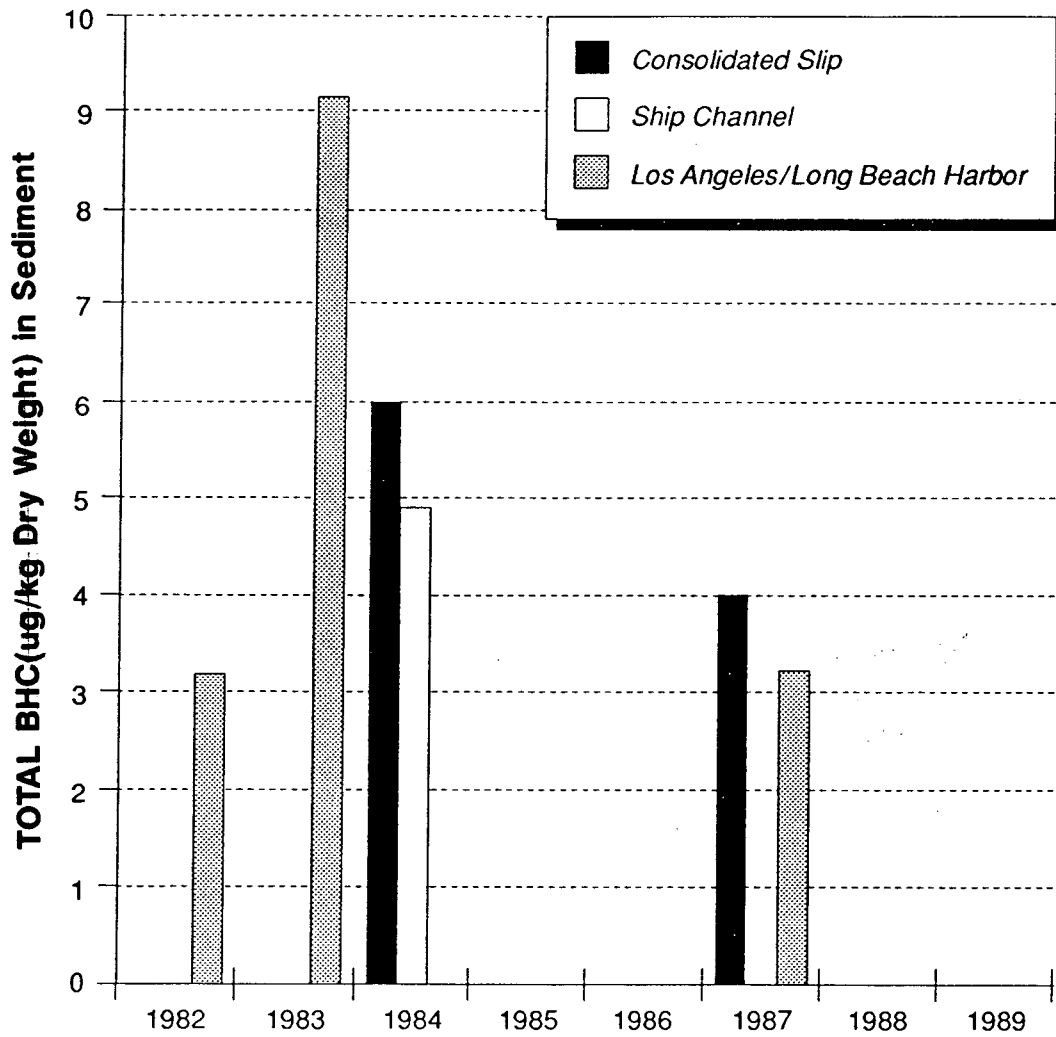


FIGURE 6-1  
 SWRCB SEDIMENT DATA  
 FOR DDT FROM CONSOLIDATED SLIP AND  
 TWO OTHER LOS ANGELES HARBOR LOCATIONS  
 Ecological Risk Assessment  
 Montrose Superfund Site



SOURCE: State Water Resources  
Control Board, undated.

FIGURE 6-2  
SWRCB SEDIMENT DATA  
FOR BHC FROM THE CONSOLIDATED SLIP AND  
TWO OTHER LOS ANGELES HARBOR LOCATIONS

Ecological Risk Assessment  
Montrose Superfund Site

## **7 Preliminary Risk Characterization**

## Section 7

# Preliminary Risk Characterization

## 7.1 Environmental Contaminant Concentrations

The following sections compare available contaminant concentration data for surface water, sediments, and soil from the study area to available criteria, and characterize risks to receptors in the study area. The risk characterization is greatly limited by the lack of available data.

### 7.1.1 Surface Water

Contaminant concentrations in surface water from 1985 to 1990, as reported in the STORET data base, are used to assess risks to aquatic receptors. Within the data base, DDT and BHC data were not available for all STORET sample locations in the Montrose drainage system. These data are, however, the only available valid surface water data. Maximum observed concentrations of DDT in surface water have exceeded marine aquatic life criteria (Table 7-1). The maximum DDT concentration for the Torrance Lateral exceeded the marine aquatic life acute criteria by nearly 3 times, and the chronic criteria by 100 times.

DDT and BHC have not been detected in the Dominguez Channel near Vermont Avenue since 1985; information on the detection limit is available. Other areas of the Channel have not been sampled. Because DDT and BHC were not detected in the Dominguez Channel only the analytical detection limits can be compared to acute and chronic exposure criteria. The detection limit for DDT was typically  $0.1 \mu\text{g/L}$ , near the acute criterion ( $0.13 \mu\text{g/L}$ ) and 100 times the chronic criterion of  $0.001 \mu\text{g/L}$  (Table 7-1). The detection limits ranged as high as  $2.0 \mu\text{g/L}$ , which exceeds the acute and chronic criteria by approximately 20 and 2,000 times, respectively. The maximum

observed BHC concentration in Dominguez Channel (0.1  $\mu\text{g/L}$ ) also was near the marine acute criterion (0.16  $\mu\text{g/L}$ ). For most of the reported results, BHC was not detected; detection limits were typically 0.05  $\mu\text{g/L}$ , below the acute and chronic criteria, but ranged to 2.0  $\mu\text{g/L}$ , above the acute and chronic criteria.

<b>Table 7-1</b> <b>STORET Maximum Waterborne Concentrations (<math>\mu\text{g/L}</math>) of DDT and BHC</b> <b>Compared to Acute and Chronic Exposure Criteria for Marine Organisms</b>						
Location	Observed	Criterion		Observed	Criterion	
	Max.	Acute	Chronic	Max.	Acute	Chronic
Torrance Lateral at Main Street	0.3 <sup>a</sup>	0.13	0.001	0.08 <sup>c</sup>	0.16	0.16 <sup>b</sup>
Dominguez Channel	<2.0 <sup>d</sup>	0.13	0.001	0.1 <sup>e</sup>	0.16	0.16 <sup>b</sup>
<sup>a</sup> Detected on 1/4/87; maximum detection limit was <2.0 on 7/22/86. <sup>b</sup> No EPA marine chronic criterion; value taken from SWRCB, 1992; see also Table 6-1. <sup>c</sup> Detected on 1/9/89; maximum detection limit was <0.5 $\mu\text{g/L}$ on 7/11/90. <sup>d</sup> At Vermont Avenue Bridge on 10/15/86; typical detection limit was <0.1 $\mu\text{g/L}$ . <sup>e</sup> Detected at "Upstream of Vermont" on 1/4/87; maximum detection limit was <2.0 $\mu\text{g/L}$ on 10/15/86, at Vermont Avenue Bridge.						

Other contaminants of concern have not been measured in surface water and, therefore, risks associated with their presence cannot be characterized.

### 7.1.2 Sediment

Maximum observed concentrations of DDT in sediments from various portions of the drainage system are shown in Table 7-2. These values cannot be evaluated directly by comparison to standards or criteria because none have been promulgated, although criteria based on carbon or organic carbon content of the sediments have been suggested (Section 6.1.1 and Table 6-2). However, only three values for TOC are available from the drainage system.

Table 7-2 Maximum Concentrations (μg/kg) of Total DDT in Sediments Compared to NOAA Effects Range Concentrations as Determined by the National Status and Trends Program			
Location and Source	Observed	Effects Levels <sup>a</sup>	
		ER-L	ER-M
Kenwood Drain			
H+A 1990	87,000	3.0	350
Torrance Lateral			
H+A 1990	1,200	3.0	350
Dominguez Channel			
H+A, 1990	13,000	3.0	350
E&E, 1991	364 <sup>b</sup>	3.0	350
Consolidated Slip			
H+A, 1990	410	3.0	350
Clark, 1982	22.7	3.0	350
<sup>a</sup> See Section 6.1.3			
<sup>b</sup> Including 84 μg DDT/kg, 130 μg DDE/kg, and 150 μg DDD/kg.			

The sediment DDT concentration value for the Consolidated Slip (from Soule and Oguri, 1980) is not considered very useful because it is from 1978. It appears likely that sediment characteristics and distribution have changed significantly since then, due to storm events and daily transport of sediments in the study area. The maximum observed DDT concentrations, as measured in sediments at the various locations, can be compared to the NOAA effects range levels for total DDT from Table 6-4 (Table 7-2). All maximum observed concentrations for DDT are far greater than the ER-L concentrations, usually by several orders of magnitude, and they also usually exceed the ER-M concentrations for total DDT.

The concentration of DDT and metabolites at two stations in the Dominguez Channel (H+A, 1990) can be normalized for organic carbon content and compared to the suggested sediment criteria (Table 7-3). Concentrations of DDT in both samples are far greater than the "safe level" for chronic exposure (Pavlou, 1987) and the mean criterion suggested by EPA (EPA, 1988). DDT concentration in sample T-6A is about

five times the level considered safe for acute exposure of aquatic life, and the concentration in sample T-9C is near the suggested safe acute limit. The DDD concentration in sample T-6A is near the maximum safe level for acute exposure, but chronic levels have not been established for DDD or DDE.

<b>Table 7-3</b> <b>Concentrations of DDT and Metabolites (mg/kg C) in Two Sediment Samples from Dominguez Channel Compared to Suggested Sediment Criteria</b>					
Chemical	Sample Concentration <sup>a</sup>		Mean Criterion <sup>b</sup>	Sediment "Safe" Level <sup>c</sup>	
	T-6A	T-9C		Acute	Chronic
DDT	118	13.3	0.828	21	0.16
DDE	71.4	16.2	--	700	--
DDD	286	50	--	325	--
<sup>a</sup> H + A, 1988. <sup>b</sup> EPA, 1988 <sup>c</sup> Pavlou, 1987					

Sediments collected from the Dominguez Channel by E&E (1991) were analyzed for DDE and DDD as well as DDT, and these three values (in  $\mu\text{g/kg}$ ) can be compared separately to the effects levels concentrations (Table 7-4):

<b>Table 7-4</b> <b>Concentrations of DDT and Metabolites (<math>\mu\text{g/L}</math>) in Dominguez Channel Sediments Compared to NS&amp;T Program Effects Levels</b>			
Chemical	Observed <sup>a</sup>	Effects Levels	
		ER-L	ER-M
p,p'-DDT	84	1.0	7.0
p,p'-DDE	130	2.0	15.0
p,p'-DDD	150	2.0	20.0
<sup>a</sup> E&E, 1991			

The observed concentrations of each contaminant were much higher than the corresponding ER-L and ER-M effects levels. Thus, based on these reported effects levels,

sediment toxicity to benthic organisms may be expected to occur in the Dominguez Channel and upstream areas nearer the Montrose site.

### 7.1.3 Soils

Concentrations of DDT in soils within 0.75 miles of the Montrose site frequently exceeded 10 mg/kg and ranged up to 98 mg/kg (see Section 3.2.2.3). Background concentrations were 1.5 mg/kg or less. Although numerical criteria for risk assessment are not available, bioaccumulation of DDT (and particularly DDE) into the terrestrial food webs can be expected to occur. For example, the average ratios of residues in earthworms to those in soil for total DDT were 5:1, and for DDE the ratio was about 10:1 (Beyer and Gish, 1980).

Other studies also have clearly demonstrated the bioaccumulation and long-term persistence of DDT and its metabolites in terrestrial ecosystems (EPA, 1975). Typical food webs in these ecosystems include terrestrial invertebrates, amphibians, reptiles, birds, and mammals. Earthworms are more tolerant of DDT than arthropods (EPA, 1975), but DDT concentrations have been correlated to earthworm mortality at a Superfund site in Massachusetts (Callahan et al., 1991). In addition, the worms containing DDT residues can serve as a significant source of exposure for terrestrial consumer organisms.

Typical degradation curves for DDT in soil show half-life values ranging from several years to a decade or more (EPA, 1975). The major DDT metabolite in soil, under normal conditions of aeration, is DDE (which tends to bioaccumulate more readily than DDT as discussed above). Long-term disappearance rates are very difficult to predict, however, because a large number of factors can affect soil persistence. While average levels of DDT are expected to decline slowly, the ratio of DDE to DDT can be expected to increase.

Half-times for reduction of DDE concentrations in earthworms were about 5.7 years following application of DDT to experimental plots and monitoring for 20 years (Beyer and Krynitsky, 1989). This estimate of persistence was considered typical for low or moderate concentrations in relatively undisturbed soils (i.e., nontilled) in temperate climates. Thus, from those investigations long-term persistence of DDT and metabolites can be expected in the study area soils.

## **7.2 Contaminant Concentrations in Biota**

No data are available for direct assessment of contaminant concentrations in biota associated with the Montrose study area. Although uptake and bioaccumulation is likely to occur in aquatic and terrestrial organisms, no data were available to assess the occurrence and the significance of contaminant concentrations in plants or animals to those organisms or their consumers.

DDT bioaccumulation in fish in the Dominguez Channel and Consolidated Slip may be estimated very generally based on the relationship developed for Los Angeles area fish by Young et al. (1991). There is a 1:1.7 ratio for sediment DDE (standardized to total organic carbon content) to fish DDT (standardized to fish lipid levels). The ratio appears to be valid over a wide concentration range for Los Angeles Harbor environment bottom fish and could be used to predict Montrose drainage area bioaccumulation effects in areas where the sediment has been adequately characterized for total organic carbon and DDT. However, only three values for TOC in sediment were available; one was for the Consolidated Slip in 1978 (Soule and Oguri, 1980), and two were for the Dominguez Channel (H+A, 1990).

The sediment TOC values were used with sediment DDT (assumed approximately equivalent to DDE) to estimate fish DDT concentrations (standardized to lipid) for the Dominguez Channel and Consolidated Slip. A fish muscle lipid level of 1 percent was

used for the standardization, as developed from fish muscle lipid levels of Los Angeles harbor fish quantified by Gossett et al. (1983).

The predicted fish muscle DDT levels ranged from 7.89 mg/kg and 1.35 mg/kg wet weight for the Dominguez Channel to 0.42 mg/kg for the Consolidated Slip. The predicted Consolidated Slip fish DDT concentration (0.42 mg/kg DDT) falls within the mid-range for Los Angeles Harbor fish muscle (0.12 to 0.83 mg/kg DDT) measured by Mearns et al. (1991), indicating the general predictive ability of the 1:1.7 sediment:fish ratio.

The predicted Dominguez Channel fish DDT values overlap with the levels measured for fish near the Los Angeles County Sanitation District marine outfalls during the 1970s (Smokler et al., 1979). These DDT levels are approximately two orders of magnitude above those of fish from Southern California island control populations (Smokler et al., 1979). Birds consuming whole fish would be potentially exposed to average DDT levels several times that estimated for fish muscle tissue because whole fish lipid levels are closer to 5 to 10 percent rather than the 1 percent found in fish muscle.

Additional estimates of potential bioaccumulation into aquatic invertebrates and fish should be possible if future sample sediment from the Dominguez Channel and Consolidated Slip are concurrently analyzed for TOC and contaminant concentrations. The EPA is conducting studies to assess the bioaccumulation potential and associated toxicity of DDT and related contaminants in sediments at a harbor site in Richmond, California (Lincoff, 1992). Those estimates, or alternatively, direct measurement of contaminant concentrations in invertebrates and fish at the Montrose site, are needed to assess risks to aquatic and semi-aquatic receptors.

DDE concentrations of only a few mg/kg in the diet of sensitive bird species can cause significant reductions in reproductive success (see Section 6 and Appendix B). Concentrations of that magnitude could be expected to occur in food-chain biota near the

Montrose property. However, the areal extent of soil DDT concentrations sufficiently elevated to cause significant bioaccumulation is not known.

Based on a DDE bioaccumulation ratio of 10:1 (earthworms:soil) earthworms living in soils in the vicinity of the Montrose property could potentially contain average DDE concentrations near 100 mg/kg in some areas (based on an approximate average of 10 mg/kg of DDE in soils, as determined from available data).

### **7.3 Toxicity Test Results**

Sediments collected from the Consolidated Slip for evaluation of a proposed dredging project (Marine Bioassay Laboratories, 1982) were toxic to several types of test organisms, including mysid shrimp, fish, sea urchins, and worms (see Section 6.4.1). However, the cause of those toxic effects was not identified and no recent (post-1982) toxicity results are available.

The EPA is conducting studies to assess toxicity of Richmond Harbor sediments to aquatic organisms (Lincoff, 1992). Because the contaminants of concern and the type of setting are the same, the findings of those studies should be useful for assessment of the Dominguez Channel and Consolidated Slip sediments. The primary organisms exposed to contaminated sediments in both the Richmond Harbor and Montrose study areas are marine/estuarine species. Results of the Richmond Harbor toxicity tests will relate effects in test organisms to the TOC and contaminant concentrations in sediments. However, because so few data for organic carbon are available for sediments in the Dominguez Channel and Consolidated Slip, and current contaminant concentrations are not known, additional sampling will be required for toxicity assessment near the Montrose property.

## 7.4 Receptor Populations

The available information indicates that aquatic, semi-aquatic, and terrestrial animals are present in areas where they are exposed to contaminated media, as described in Sections 3 and 4. Thus, exposure pathways for ecological receptors (described in Section 5) are expected to be complete; however, current levels of exposure are not known because recent sediment and biota sampling have not been conducted).

Within portions of the surface drainage pathway, the most significant exposures for aquatic organisms are expected to be at the confluence of the Torrance Lateral with the Dominguez Channel and in the Consolidated Slip. Within those areas, the greatest exposures to receptor organisms can be expected from accumulated sediments containing DDT and its metabolites.

Previous studies have provided limited diversity or abundance information about fish and macro-invertebrates populations in lower portions of the Dominguez Channel and in the Consolidated Slip (Section 4.2). Although the aquatic fauna in the Dominguez Channel near the Torrance Lateral have not been characterized, fish and benthic invertebrates are expected to be present. Semi-aquatic birds that feed on those organisms were observed foraging in this area during the reconnaissance surveys (especially in February). These birds' behavior indicates that food organisms are present. Similarly, the presence and foraging behavior of semi-aquatic birds in the Consolidated Slip indicate that fish and aquatic invertebrates do also occur there, although species composition is unknown in detail. All of the semi-aquatic bird species observed in the Dominguez Channel are protected by the Migratory Bird Treaty Act, and the cormorant is a California species of special concern.

No information was available concerning terrestrial invertebrates in downwind areas from the site, and those animals were not surveyed during the site visits. However, earthworms and arthropods (insects, spiders, etc.) are widely distributed, and these

organisms can be expected to bioaccumulate DDT and its metabolites in that ecosystem. Ingestion of terrestrial organisms or soil would result in exposure of vertebrates (amphibians, reptiles, birds, and mammals) living in the vicinity of the site. The extent of bioaccumulation of contaminants by terrestrial invertebrates is not known, although those living within 0.75 mile of the site could be expected to contain DDT and metabolites at potentially harmful concentrations (see 7.1 above). Soil contamination levels beyond 0.75 mile are not known.

Aside from limited studies conducted near the ARCO Watson Refinery and in the Consolidated Slip (Section 4.2), there have been no measurements of aquatic or terrestrial biological community structure in the drainage system or downwind areas of the study area. Effects on community structure are most likely to occur in the areas of sediment accumulation because the sediments appear to serve as a reservoir of those contaminants that adsorb to soil and sediment particles.

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## 8 Conclusions and Limitations

## Section 8

# Conclusions and Limitations

## 8.1 Conclusions

The available information leads to several conclusions concerning risks to ecological receptors in the study area:

- The Montrose property is a source of contamination for downstream and downwind areas, particularly for DDT and its metabolites.
- Chemicals of greatest concern are those that a) persist in the soils and sediments in the study area, b) are toxic at concentrations found in those media or in surface water, and c) tend to bioaccumulate in animals exposed to them. Available data indicate that DDT and its metabolites are the primary chemicals of ecological concern.
- Ecological receptors in downstream areas (primarily the Dominguez Channel and Consolidated Slip) include aquatic invertebrates, fish, and semi-aquatic birds. These species are exposed to contaminants in the sediments and surface water through ingestion and dermal contact that can result in toxic effects and bioaccumulation of chemicals.
- Waterborne concentrations of DDT have exceeded water quality criteria in the Torrance Lateral. Maximum observed concentrations of DDT and BHC in the Dominguez Channel and Consolidated Slip cannot be evaluated because the detection limits were at or above the acute and chronic criterion for those chemicals (and because water solubility is very low).

- Concentrations of DDT, DDE, and DDD in sediments at the intersection of the Dominguez Channel and Torrance Lateral exceed levels associated with reported adverse effects in biota or those that have been suggested as sediment criteria.
- Contaminant concentrations have not been measured in biota from the surface drainage systems, but bioaccumulation of DDT and metabolites by aquatic invertebrates and fish is expected to reach levels causing adverse effects in fish and birds consuming them. Bioaccumulation of other chemicals is not expected to be significant due to their lower soil affinity.
- Concentrations of DDT in surface soils within 0.75 miles of the Montrose property frequently exceeded 10 mg/kg. Although numerical criteria for evaluation of these concentrations are not available, bioaccumulation of DDT (and particularly DDE) into the terrestrial food webs can be expected to occur. A bioaccumulation ratio of 10:1 can be expected for earthworms living in DDE-contaminated soils.
- Long-term persistence of DDT and metabolites can be expected in soils and sediments.

## 8.2 Limitations

The data on the nature and extent of contamination by the Montrose chemicals of concern and the information on present populations of receptor organisms in the Dominguez Channel, the Consolidated Slip, and downwind areas from the site are

inadequate to assess the present ecological risk. Lack of current information on the following topics limits the completeness of the risk assessment:

- Current distribution and chemical characteristics of surface water and sediments in the Kenwood Drain, Torrance Lateral, Dominguez Channel, and Consolidated Slip are not known. Heavy rainfall and consequent stormwater flows through the Dominguez Channel may have greatly altered areas of sediment accumulation. There is very limited information on TOC content of the sediments (only two samples in the recent past), and TOC information is needed to predict toxicity and bioaccumulation. Contaminant profiles in the sediments also could have been altered by stormwater flows during the most recent winter (1992) storms.
- Aquatic communities present in the Dominguez Channel, in particular, are incompletely characterized, and they have not been studied for over 15 years. Food web linkages have not been delineated for any of the aquatic environments in the Montrose study area. Instead, linkages were inferred from Los Angeles Harbor studies. Similarly, terrestrial receptors have not been surveyed in sufficient detail to characterize their exposure risks. At present, it is impossible to accurately assign levels of risks for the invertebrates, fish, amphibians, birds, and mammals of these environments.
- Sediment toxicity information is lacking for the Dominguez Channel sediment and needs to be updated for the Consolidated Slip to assess current conditions.
- Recent bioaccumulation data are lacking for all Montrose chemicals of concern, especially in the areas most likely to be affected. Available data suggest that significant bioaccumulation can be expected, but the data are

inadequate for estimating concentrations and exposures in aquatic or terrestrial areas.

- Waterborne concentrations of contaminants of concern (especially DDT and metabolites plus BHC) have not been well characterized. STORET data base results were highly variable and sample locations limited. Concentrations may also be different than those measured 5 years or longer ago.
- It is unclear whether acetone should be considered a contaminant of concern or a laboratory contaminant. Acetone would be a contaminant of ecological concern:
  - If it is present in sediments at concentrations that would affect solubility/availability of other contaminants.
  - If concentrations in water, soil/sediment, or air could be toxic to ecological receptors. However, current data are insufficient to conclude whether it is a contaminant of concern.

## **9 Recommendations for Further Studies**

## Section 9

### Recommendations for Further Studies

The following studies are recommended to address the limitations of the risk assessment:

- Distribution of sediments within the surface drainage system should be surveyed to determine current distribution patterns.
- Sediments should be concurrently analyzed for contaminants of concern, grain size, and TOC content. This data may be adequate for assessment of possible toxicity to aquatic organisms, using relationships being developed by EPA for Richmond Harbor (Lincoff, 1992). Special care should be taken to determine whether acetone occurs in sediments.
- Uptake and bioaccumulation of DDT and its metabolites in aquatic organisms should be assessed through the tissue analysis of benthic invertebrates and fish (especially benthic-feeding and demersal species, if present) in the Dominguez Channel. Resident species (i.e., less mobile) would be the preferred indicator organisms. The area of greatest need for sampling is in the vicinity of Torrance Lateral. Contaminant concentrations should be compared to values predicted using the Richmond Harbor relationships.
- Bioaccumulation studies with mussels should continue in the Consolidated Slip and should be supplemented by measurements from several locations in the Dominguez Channel.

- The aquatic communities of the Dominguez Channel and Consolidated Slip should be studied over a 1-year period to identify population levels, seasonal trends, and food web linkages, particularly linkages that lead to aquatic birds. Our initial assessment of toxicity to aquatic organisms in the Montrose drainage system is based on established toxicity information for marine organisms and the estimated degree of exposure of the local populations. Exposure pathways are estimated based on a general knowledge of the food web and species observed during two field surveys. The pathways of most concern are those from sessile, detritivorous, and in-faunal or epibenthic invertebrates, such as are found in the Dominguez Channel and Consolidated Slip, to predatory invertebrates or fish, and eventually from either of those two routes to larger fish or birds as functional top predators. The critical exposure route for the chemicals of concern is through the sediment and resuspended sediment in the water column to deposit, suspension, or filter feeders. Figures 5-1 and 5-2 summarize the conceptual model of exposure for organisms in the Dominguez Channel and Consolidated Slip. Actual food webs of the populations and studies of contaminant transfer specific to these areas have not yet been established by field observation and collections.
- Concentrations of contaminants of concern should be measured in surface water during dry-season and wet-season periods, especially in the areas where ambient water quality criteria were exceeded. Special care should be used to determine whether acetone occurs in the water or it is an analytical laboratory contaminant.
- Concentrations of DDT and its metabolites should be measured in earthworms from areas where elevated concentrations were found in surface soils (within a 1-mile radius of the Montrose property) and higher trophic level consumers may be exposed by consuming the worms. Toxic effects

of the contaminants of concern should be tested with earthworms, as described by Callahan et al. (1991).

- DDT and metabolites should be measured in near-surface soils in open areas farther downwind from the Montrose property (such as the Dominguez Golf Course) to assess the potential significance of bioaccumulation by earthworms (and transfer through terrestrial food webs).

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## 10 References Cited

## Section 10

### References Cited

Acronyms that were abbreviated in text include the following. They are alphabetized by their abbreviated letters not by their full names:

ASTER	Assessment Tools for the Evaluation of Risk Ecotoxicity Profiles
ATSDR	Agency for Toxic Substances and Disease Registry
BELA	Biomedical and Environmental Information Analysis
CDFG	California Department of Fish and Game
CEC	California Energy Commission
DHEW	Department of Health, Education, and Welfare
DOJ	Department of Justice
E&E	Ecology and Environment
EPA	Environmental Protection Agency
H+A	Hargis + Associates, Inc.
HSDB	Hazardous Substances Data Bank
IARC	International Agency for Research on Cancer
LACDPW	Los Angeles County Department of Public Works
LACFCD	Los Angeles County Flood Control District
M&E	Metcalf & Eddy, Inc.
MEC	MEC Analytical Systems, Inc.
NIOSH	National Institute for Occupational Safety and Health
NOAA	National Oceanic and Atmospheric Administration
NRC	National Research Council
NTP	National Toxicology Program
POLA	Port of Los Angeles
RTECS	Registry of Toxic Effects of Chemical Substances
SWRCB	State Water Resources Control Board
WHO	World Health Organization

Anderson, D.W., and J.J. Hickey. 1972. Eggshell changes in certain North American birds. Proc. Int. Ornith. Congr. 15:514-540.

Anderson, D.W. et al. 1975. Brown Pelicans: Improved Reproduction off the Southern California Coast. Science. Vol. 190. pp 806-808.

Anderson, D.W., R.M. Jurek, and J.O. Keith. 1977. The Status of Brown Pelicans at Anacapa Island in 1975. California Fish and Game. Vol. 63. pp 4-10.

Andrew et al. 1981. As cited in ATSDR, 1990b.

API. 1981. As cited in ATSDR, 1989c.

ASTER Ecotoxicity Profile. 1992. EPA Environmental Research Laboratory. Duluth.

Atlas, E. et al. 1982. Air-sea exchange of high molecular weight organic pollutants: laboratory studies. Environ. Sci. Technol. 16:283-286.

ATSDR. 1987.

\_\_\_\_\_. 1989a. Toxicology Profile for Chloroform. National Technical Information Service PB89-160360. January.

\_\_\_\_\_. 1989b. Toxicology Profile for Benzene. National Technical Information Service PB89-209464. May.

\_\_\_\_\_. 1989c. Toxicological Profile for DDT, DDE, and DDD. U.S. Public Health Service Contract No. 205-88-0608. December.

\_\_\_\_\_. 1989d. Toxicology Profile for 1,2-Dichloroethane. National Technical Information Service PB90-171422. December.

\_\_\_\_\_. 1989e. Toxicological Profile for Toluene. U.S. Environmental Protection Agency, Washington, D.C., National Technical Information Service PB90-198904. December.

\_\_\_\_\_. 1989f. Alpha-, Beta-, Gamma-, and Delta-Hexachlorocyclohexane. National Technical Information Service PB90-171406. December.

\_\_\_\_\_. 1990a. Toxicological Profile for Chlorobenzene. U.S. Department of Health and Human Services, Washington, D.C., National Technical Information Service PB91-180505. December.

\_\_\_\_\_. 1990b. Toxicology Profile for Ethylbenzene. U.S. Department of Health and Human Services, Washington, D.C., National Technical Information Service PB91-180372. December.

\_\_\_\_\_. 1990c. Toxicological Profile for Total Xylenes. National Technical Information Service PB91-181552. December.

Bailey, S. et al. 1969a. The metabolism of 1,1-di(p-chlorophenyl)-2,2,2-trichloroethane and 1,1-di(p-chlorophenyl)-2,2-dichloroethane in the pigeon. *Toxicol. Appl. Pharmacol.* 14:13-22.

Bailey, S. et al. 1969b. The metabolism of 1,1-di(p-chlorophenyl)-2,2-dichloroethylene and 1,1-di(p-chlorophenyl)-2-chloroethylene in the pigeon. *Toxicol. Appl. Pharmacol.* 14:23-22.

Ballou, T.G., et al. 1985. Summary and evaluation of THR Toxicological and physiological effects of pollutants on shellfish. Part Two (Petroleum Hydrocarbons) and Part Three (Pesticides). NOAA, Office of Marine Pollution Assessment, Office of Operational Programs RD/MP2. Seattle, Washington.

BEIA. 1989. The Installation Restoration Program Toxicology Guide. Volumes I-IV. BEIA Health and Safety Research Division, Oak Ridge National Laboratory. July.

Beyer, W.N., and C.D. Gish. 1980. Persistence in earthworms and potential hazards to birds of soil applied DDT, dieldrin, and heptachlor. *J. App. Ecol.* 17:295-307.

Beyer, W.N., and A.J. Krynitsky. 1989. Long-term persistence of dieldrin, DDT, and heptachlor epoxide in earthworms. *AMBIO*, 18:271-273.

Beyer, W.N. 1990. Evaluating Soil Contamination. Biological Report 90(2). U.S., Fish and Wildlife Service. Washington, D.C. July.

Beyer, N., E. Connor, and S. Gerould. 1992. Survey of Soil Ingestion by Wildlife. Manuscript in review. U.S. Fish and Wildlife Service.

Blakley, B.R. 1982. Lindane toxicity in pigeons. *Can. Vet. J.* 23:267-268.

Blus, L.J., R.G. Heath, C.D. Gish, A.A. Belisle, and R.M. Prouty. 1971. Eggshell thinning in the brown pelican: implication of DDE. *BioScience* 21 (24):1213-1215.

Blus, L.J., C.D. Gish, A.A. Belisle, and R.M. Prouty. 1972a. Logarithmic relationship of DDE residues to eggshell thinning. *Nature* 235 (5338):376-377.

Blus, L.J., C.D. Gish, A.A. Belisle, and R.M. Prouty. 1972b. Further analysis of the logarithmic relationship of DDE residues to eggshell thinning. *Nature* 240 (5377):164-166.

Blus, L.J. 1970. Measurements of brown pelican eggshells from Florida and South Carolina. *BioScience* 20 (15):867-869.

Blus, L.J., A.A. Belisle, and R.M. Prouty. 1974a. Relations of the brown pelican to certain environmental pollutants. *Pestic. Monit. J.* 7 (3/4):181-194.

Blus, L.J., B.S. Neely, Jr., A.A. Belisle, and R.M. Prouty. 1974b. Organochlorine residues in brown pelican eggs: relation to reproductive success. *Environ. Pollut.* 7 (2):81-91.

Blus et al. 1984. Effects of heptachlor- and lindane-treated seed on Canada geese. *J. Wildl. Manage.* 48:1097-1111.

Buikema, A.L., Jr., and A.C. Hendricks. 1980. Benzene, Xylene, and Toluene in Aquatic Systems: A Review. Washington, D.C.: American Petroleum Institute.

Burnett, R. 1971. DDT residues: Distribution of concentrations in *Emerita analoga* (Stimpson) along coastal California. *Science* 174:606-608.

Burrage, R.H., and J.G. Saha. 1972. Insecticide residues in pheasants after being fed on wheat seed treated with heptachlor and <sup>14</sup>C-lindane. *J. Econ. Entomol.* 65:1013-1017.

California Mussel Watch Data. Undated. Preliminary results for 1987-88, 1988-89, and 1989-90 sampling. Division of Water Quality, State Water Resources Control Board. Sacramento, California.

CDFG. 1951. Los Angeles-Long Beach Harbor Pollution Study, S19999, Project No. 5246. (DOJ, SFG 009, 2316-2331)

\_\_\_\_\_. 1989. Wildlife Habitat Relationships. Sacramento, California.

\_\_\_\_\_. 1991. RAREFIND: Unpublished Natural Diversity Database Report. California Department of Fish and Game, Sacramento, California.

Caldwell and Mallon. 1977. As cited in Ballou et al., 1985.

Callahan et al. 1979. As cited in HSDB, 1992.

Callahan et al. 1979. As cited in BEIA, 1989.

Callahan, et al. 1991. Onsite Methods for Assessing Chemical Impact on the Soil Environment Using Earthworms: A Case Study at the Baird and McGuire Superfund Site, Holbrook, Massachusetts. *Environmental Toxicology and Chemistry* 10:817-826.

Cantelmo et al. 1982. As cited in Ballou et al., 1985.

CEC. 1986. Biological Resources, Testimony of Ralph Appy. CEC-SA-6077-D-801, January 8, 9.

CH2M HILL. 1992. Work Scope for Ecological Risk Assessment, Montrose Chemical Corporation, Torrance, California. CH2M HILL. March 18.

Chiou et al. 1982. As cited in BEIA, 1989.

Clark, Earl K. 1982. Consolidated Slip Dredging Project (In The Port of Los Angeles, Graphs, Maps, and Memo included in Document, Attachments 1-18 included). November 8 (DOJ, ACE024, 0405-0480).

Clarke. 1981. As cited in HSDB, 1992.

Clayton, G.D., and F.E. Clayton (eds). 1981. *Patty's Industrial Hygiene and Toxicology*, Third Revised Edition. Volumes 2A, 2B, Toxicology. New York: John Wiley and Sons, Inc.

Clayton. 1981. As cited in BEIA, 1989.

Cooke, A.S. 1973. Shell thinning in avian eggs by environmental pollutants. *Environ. Pollut.* 4:85-152.

Cragg et al. 1989. As cited in ASTDR, 1990b.

Courtney, K.D. et al. 1986. A perinatal study of toluene in CD-1 mice. *Fund. Appl. Toxicol.* 6:145-154.

Darrow, D.C., and C.H. Harding. 1975. Accumulation and apparent absence of DDT metabolism by marine copepods, *Calanus* spp., in culture. *J. Fish. Res. Board Can.* 32:1845-1849.

Davison, K.L., and J.L. Sell. 1974. DDT thins shells of eggs from mallard ducks maintained on *ad libitum* or controlled-feeding regimens. *Arch. Environ. Contam. Toxicol.* 2:222-232.

Deichman, W.B. 1981. Halogenated cyclic hydrocarbons. Pages 3604-3761 in *Patty's Industrial Hygiene and Toxicology*, edited by G.D. Clayton and F.E. Clayton. New York: John Wiley and Sons.

DeWaziers, I., and V. Azias. 1987. Drug-metabolizing enzyme activities in the liver and intestine of rats exposed to DDT: Effects of Vitamin A status. *Arch. Environ. Contam. Toxicol.* 16:343-348.

Dieter, M.P. 1975. Further studies on the use of enzyme profiles to monitor residue accumulation in wildlife: plasma enzymes in starlings fed graded concentration of mor-sodren, DDE, aroclor 1254, and malathion. *Arch. Environ. Contam. Toxicol.* 3:142-150.

Dieter, M.P. 1974. Plasma enzyme activities in coturnix quail fed graded doses of DDE, polychlorinated biphenyl, malathion and mercuric chloride. *Toxicol. Appl. Pharmacol.* 27:86-98.

DOJ. Issues Relating to Federally Permitted Releases Under CERCLA, Montrose Chemical and Levin Richmond Sites. DOJ No. NFS011, 1682-1684.

E&E. 1986. Amended CERCLA Site Sampling Plan and Sampling Documentation Report—Offsite Sample for Air Dispersion of DDT, Montrose Chemical Corporation. October 17, 1986.

\_\_\_\_\_. 1991. Listing Site Inspection Summary Report. October 30.

Eberhardt et al. 1971. As cited in HSDB, 1992.

Envirologic Data, Inc. 1990. Draft Risk Assessment for Off-Property Surficial Soil at the Montrose Site, Torrance, California. November 30.

\_\_\_\_\_. Draft Health and Environmental Evaluation, Montrose Site, Torrance, California, Volumes I-III. April 10.

EPA. 1975. DDT—A Review of Scientific and Economic Aspects of the Decision to Ban Its Use as a Pesticide. EPA/540/1-75-022. July.

\_\_\_\_\_. 1980a. As cited in BEIA, 1989.

\_\_\_\_\_. 1980b. As cited in BEIA, 1989.

\_\_\_\_\_. 1980c. As cited in BEIA, 1989.

\_\_\_\_\_. 1980d. As cited in BEIA, 1989.

\_\_\_\_\_. 1980e. As cited in BEIA, 1989.

\_\_\_\_\_. 1980f. As cited in BEIA, 1989.

\_\_\_\_\_. 1980g. As cited in BEIA, 1989.

\_\_\_\_\_. 1988. Interim Sediment Criteria Values for Nonpolar Hydrophobic Organic Contaminants. SCD#17. May.

\_\_\_\_\_. 1989. Risk Assessment Guidance for Superfund, Volume II, Environmental Evaluation Manual, Interim Final. EPA/540/1-89/001. March.

\_\_\_\_\_. 1986. Quality Criteria for Water. EPA 440/5-86-001. Washington, D.C. May 1.

\_\_\_\_\_. 1992. ECO Update: Developing a Work Scope for Ecological Assessments. Intermittent Bulletin Vol. 1, No. 4. Publication 9345.0-05I. May.

Faber, R.A., and J.J. Hickey. 1973. Eggshell thinning, chlorinated hydrocarbons, and mercury in inland aquatic bird eggs, 1969 and 1970. *Pestic. Monit. J.* 7:27-36.

Faber et al. 1972. Organochlorines and mercury in common egrets and great blue herons. *Environ. Pollut.* 3:111-122.

Fabro et al. 1984. As cited in ATSDR, 1989c.

Ferrando, M.D. et al. 1989. Short-term lindane effects on gill tissue metabolism of the eel. *Toxicol. Environ. Chem.* 24:17-23.

Ferrando, M.D. and Andreu-Moliner. 1991. Acute lethal toxicity of some pesticides to *Brachionus calyciflorus* and *Brachionus plicatilis*. *Bull. Environ. Contam. Toxicol.* 47:479-484.

Friend, M., M.A. Haegele, and R. Wilson. 1973. DDE: Interference with extrarenal salt excretion in the mallard. *Bull. Environ. Contam. Toxicol.* 9:49-53.

Fry, D.M., and C.K. Toone. 1981. DDT-induced feminization of gull embryos. *Science* 213:922-924.

Fry, D.M. et al. 1987. Sex ratio skew and breeding patterns of gulls: demographic and toxicological considerations. *Studies in Avian Biology* 10:26-43.

Gellert, R.J. et al. 1974. Effects of neonatally administered DDT homologues on reproductive function in male and female rats. *Neuroendocrinology* 16:84-94.

Gish, C.D., and N.J. Chura. 1970. Toxicity of DDT to Japanese quail as influenced by body weight, breeding condition, and sex. *Toxicol. Appl. Pharmacol.* 17 :740-751.

Goldman, C.R., and A.J. Horne. 1983. *Limnology*. New York: McGraw-Hill Book Co.

Gosselin. 1984. As cited in HSDB, 1992.

Gossett, R.W. et al. 1983. DDT, PCB, and benzo(a)pyrene levels in white croaker (*Genyonemus lineatus*) from southern California. *Mar. Pollut. Bull.* 14:60-65.

Grant, W.M. 1974. *Toxicology of the Eye*. Second Edition. Springfield, Illinois: Charles C. Thomas Publisher.

Greichus, Y.A., and M.R. Hannon. 1973. Distribution and biochemical effects of DDT, DDD and DDE in penned double-crested cormorants. *Toxicol. Appl. Pharmacol.* 26:483-494.

Gress et al. 1973. Reproductive failures of double-crested cormorants in southern California and Baja California. *Wilson Bull.* 85:197-208.

H+A. 1990. Preliminary Draft Remedial Investigation, Montrose Site, Torrance, California. October 31. Volumes I-IV.

Haegerle, M.A., and R.H. Hudson. 1974. Eggshell thinning and residues in mallards one year after DDE exposure. *Arch. Environ. Contam. Toxicol.* 2:356-363.

Hardin et al. 1981. As cited in BEIA, 1989.

Hart et al. 1972. As cited in ATSDR, 1989c.

Heath, R.G., J.W. Spann, and J.F. Kreitzer. 1969. Marked DDE impairment of mallard reproduction in controlled studies. *Nature* 224 (5214):47-48.

Heinz, G. 1976. Behavior of mallard ducklings from parents fed 3 ppm DDE. *Bull. Environ. Contam. Toxicol.* 16:640-645.

Heppel, L.A. et al. 1946. Toxicology of 1,2-dichloroethane; V. effects of daily inhalations. *J. Ind. Hyg. Toxicol.* 28:113-120.

Hertel, Raymond M. 1966. Letter to State of California Regional Water Pollution Control Board No. 4, Los Angeles Region, Adopting Long Range Waste Disposal and Water Quality Objectives for the Dominguez Flood Control Channel from Raymond Hertel, Los Angeles Regional Water Quality Control Board; November 3. (DOJ-MWB005, 1741-1745).

Hertel, R.M. 1969. Review of Information Pertinent to Los Angeles—Long Beach Harbor and Dominguez Channel. May, 1969. pp. I.1—VIII.10. A staff Report to the Los Angeles Regional Water Quality Control Board. State of California. The Resources Agency. (DOJ, SFG001, 0977-1070).

Hickey, J.J., and D.W. Anderson. 1968. Chlorinated hydrocarbons and eggshell changes in raptorial and fish-eating birds. *Science* 162 (3850):271-273.

Hill, L.R. 1974. A Seasonal Environmental Study of the Polychaetous Annelids Within the Long Beach Naval Station and Shipyard, Long Beach, California. Master's Thesis. California State University, Long Beach, California.

Hill E.F., and M.B. Camardese. 1986. Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to Coturnix. U.S. Fish and Wildlife Service, Fish and Wildlife Technical Report 2, Washington, D.C.

Hill, E.F. et al. 1975. Lethal Dietary Toxicities of Environmental Pollutants to Birds. U.S. Fish and Wildlife Service. Special Scientific Report-Wildlife No. 191. Washington, D.C.

Hodgson, E., and P.E. Levi. 1987. *A Textbook of Modern Toxicology*. New York: Elsevier Science Publishing Co., Inc.

Howard et. al. 1991. *Handbook of Environmental Degradation Rates*. Chelsea, Michigan: Lewis Publishers, Inc.

HSDB. 1992. Online through TOXNET System of National Library of Medicine, accessed April 22.

Hudak and Ungvary. 1978. As cited in ATSDR, 1990c.

Hudson, R.H. et al. 1984. *Handbook of Toxicity of Pesticides to Wildlife*. Second Edition. U.S. Dept. of Interior. Fish and Wildlife Service. Res. Pub. 153. Washington, D.C.

IARC. 1983. Working Group on the Evaluation of the Carcinogenic Risk of Chemicals to Man. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 29. Geneva: World Health Organization.

Jarvis. 1975. As cited in CEC, 1986.

Jefferies, D.J. 1971. Some sublethal effects of p,p'-DDT and its metabolites p,p'-DDE on breeding passerine birds. *Med. Fak. Landb. Wentenschappen Gent*. 36:34-42.

John et al. 1984. As cited in ATSDR, 1990a.

Johnson, Norman S. 1970. Letter to the Honorable Board of Supervisors, from Norman Johnson, Director, County of Los Angeles, Department of Parks and Recreation. October 14, 1970.

Kadeg et al. 1986. As cited in BEIA, 1989.

Klaassen, C.D., M.O. Amdur, and J. Doull (eds). 1986. *Casarett and Doull's Toxicology, The Basic Science of Poisons*. Third Edition. New York: Macmillan Publishing Company.

Kluwe, W.M. et al. 1985. Toxic responses to acute, subchronic, and chronic oral administration of monochlorobenzene to rodents. *J. Toxicol. Environ. Health* 15:745-767.

Kreitzer, J.F., and G.H. Heinz. 1974. The effect of sublethal dosages of five pesticides and a polychlorinated biphenyl on the avoidance response of coturnix quail chicks. *Environ. Pollut.* 6:21-29.

LACPPW. 1991. Drains and Facilities, Los Angeles County, Thomas Guide. Thomas Bros. Maps. Irvine, CA.

LACFCD. 1967. Disposal of Wastes into the Pacific Ocean, Los Angeles and Long Beach Harbor Area and Dominguez Channel. Los Angeles Department of County Engineers, County Sanitation District. (SFG081, 1406-1428).

LACFCD. 1973.

Lee, H.M., and W.J. Vissek. 1975. Sexual Differentiation of the Brain in Male Rats Given o,p'-DDT at Birth. Fed. Proc. 34:246. Abstract.

Leffler. 1975. As cited in Ballou et al., 1985.

Legone. 1974. As cited in Ballou et al., 1985.

Leo. 1983. As cited in BEIA, 1989.

Lincoff, A. 1992. Personal correspondence between Andrew Lincoff/EPA and Susan Keydel/CH2M HILL. July 30.

Long, E.R., and L.G. Morgan. 1990. The Potential for Biological Effects of Sediment-Sorbed Contaminants Tested in the National Status and Trends Program. Technical Memorandum NOS OMA 52. Seattle, Washington. March.

Long Beach and Los Angeles Harbor Departments and the U.S. Army Corps of Engineers, Los Angeles District. 1990. Phase I—2020 Plan and Feasibility Study.

Longcore, J.R. et al. 1971a. Changes in mineral composition of eggshells from black ducks and mallards fed DDE in the diet. Bull. Environ. Contam. Toxicol. 6:345-350.

Longcore, J.R. et al. 1971b. DDE thins eggshells and lowers reproductive success of captive black ducks. Bull. Environ. Contam. Toxicol. 6:485-490.

Longcore, J.R., and F.B. Samson. 1973. Eggshell breakage by incubating black ducks fed DDE. J. Wildl. Manage. 37:390-394.

Lowe, J.I. et al. 1971. Chronic exposure of oysters to DDT, toxaphene and parathion. Proc. Nat. Shellfisheries Assoc. 61:71-79.

Ludke, J.L. 1974. Interaction of dieldrin and DDE residues in Japanese quail (*Coturnix coturnix japonica*). Bull. Environ. Contam. Toxicol. 11:297-302.

Lyman. 1985. As cited in BEIA, 1989.

Lyman and Reehl. 1982. As cited in BEIA, 1989.

M&E. 1986. Draft Preliminary Report: Remedial Investigation, Part I. Montrose Facility Site, Los Angeles, California. March.

MacKay and Shiu. 1981. As cited in BEIA. 1989.

Mackison et al. 1981. As cited in BEIA, 1989.

Mahoney, J.J. 1975. DDT and DDE effects on migratory condition in white-throated sparrows. J. Wildl. Manage. 39:520-527.

Marine Bioassay Laboratories. 1982. Technical Evaluation of Environmental Impact Potential for Proposed Ocean Disposal of Dredged Material from Los Angeles Harbor, Capital Projects. Part IV, Consolidated Slip (Berth 200Y). Prepared for: Los Angeles Harbor Department. pp. 1-42 (DOJ, MEP003, 1167-1220). June.

Marks, T.A., T.A. Ledoux, and J.A. Moore. 1982. Teratogenicity of a commercial xylene mixture in the mouse. J. Toxicol. Environ. Health 9:97-105.

Matsumura, F. 1985. *Toxicology of Insecticides*. Second Edition. New York: Plenum Press.

Maybe et al. 1981. As cited in BEIA, 1989.

Mayer, K.E., and W.E. Laudenslayer, Jr. (eds). 1988. *A Guide to Wildlife Habitats of California*. California Department of Forestry and Fire Protection, Sacramento, California.

McLane, M.A.R., and L.C. Hall. 1992. DDE thins screech owl eggshells. Bull. Environ. Contam. Toxicol. 8:65-68.

McLeese and Metcalfe. 1980. As cited in ASTER, 1992.

Means et al. 1982. As cited in BEIA, 1989.

Mearns, A.J. et al. 1991. Contaminant Trends in the Southern California Bight: Inventory and Assessment. NOAA Technical Memorandum NOS ORCA 62. Seattle, Washington.

MEC. 1988. Biological Baseline and Ecological Evaluation of Existing Habitats in Los Angeles Harbor and Adjacent Waters. Prepared for Port of Los Angeles, Environmental Management Division, San Pedro, California, MEC Analytical Systems, Inc., Carlsbad, California.

Meister, R.T., ed. 1989. *Farm Chemicals Handbook*. Volume 75. Meister Publishing Co.

Meister, R.T., ed. 1990. *Farm Chemicals Handbook*. Volume 76. Meister Publishing Co.

Meith-Avcin, N. 1974. DDT and the rugophilic response of settling barnacles *Balanus improvisus*. J. Fish. Res. Board Can. 31 (12):1960-1963.

Mendenhall, V.M., E.E. Klaas, and M.A.R. McLane. 1983. Breeding success of barn owls (*Tyto alba*) fed low levels of DDE and dieldrin. Arch. Environ. Contam. Toxicol. 12:235-240.

Miller, D.S. et al. 1976. DDE feeding and plasma osmoregulation in ducks, guillemots and puffins. American Journal of Physiology, 231 (2):370.

Miller, D.J., and R.N. Lea. 1972. Guide to the Coastal Marine Fishes at California. Calif. Dept. Fish, Game Fish. Bull. No. 157.

Montgomery Research, Inc. 1967. Statement to the LA-RWQCB by the Dominguez Channel Users Group Concerning the Establishment of Water Quality Standards for Dominguez Channel and the Los Angeles-Long Beach Inner Harbor. January. (MWB005 1303-1340).

Monsanto Company. 1978. Industrial Bio-Test Draft Report of 90-Day Subacute Vapor Inhalation Toxicity Study with Monochlorobenzene in Beagle Dogs and Albino Rats. TSCA Sec 8 (d) Submission 8DHQ-1078-0212 (1).

Montrose. 1981. Montrose Chemical Corporation of California Inter-Office Correspondence From John L. Kallok to R.G. Wilson. May 21. (DOJ No. NFS043, 0656-0657).

Morrow. 1974. As cited in Verschueren, 1983.

Nair, R.S. et al. 1987. A two-generation reproduction study with monochlorobenzene vapor in rats. Fundam. Appl. Toxicol. 9:678-686.

Nakahara, E. 1992. Personal communication with Eiji Nakahara, Imperial Department of Public Works, Los Angeles County, and Susan Keydel/CH2M HILL. July 1.

Nawrot, P.S., and R.E. Staples. 1980. Embryofetal toxicity and teratogenicity of isomers of xylene in the mouse. Soc. Toxicol. Abst. Pap. 19th 1980:AZZ.

Nawrot, P.S., and R.E. Staples. 1979. Embryo-fetal toxicity and teratogenicity of benzene and toluene in the mouse. Teratology 19:41A.

Neely et al. 1985. As cited in BEIA, 1989.

NIOSH. 1973. Criteria for a Recommended Standard... Occupational Exposure to Toluene. DHEW Publication No. (HSM) 73-11023.

\_\_\_\_\_. 1978. Special Occupation Hazard Review for DDT. DHEW (NIOSH) Publication No. 78-200.

\_\_\_\_\_. 1989. Registry of Toxic Effects of Chemical Substances. Online file, January.

NOAA. 1988. Preassessment Screen Determination Case No. UD No. 2 IX-6:16-54.

\_\_\_\_\_. 1990. Damage Assessment Plan, Los Angeles and Long Beach Harbors, Palos Verdes Shelf, and Ocean Dump Sites. February 6, 1990.

\_\_\_\_\_. 1991. Draft Injury Determination Plan. Damage Assessment: Los Angeles/Long Beach Harbors, Palos Verdes Shelf, and Ocean Dump Sites. National Oceanic and Atmospheric Administration, U.S. Department of Commerce, U.S. Department of Interior, State of California. March 8, 1991.

NRC Committee on Alkyl Benzene Derivatives. 1981. The Alkyl Benzenes. Washington, D.C.: National Academy Press.

NTP. 1986. Toxicology and Carcinogenesis Studies of Benzene (CAS No. 71-43-2) in F344/N Rats and B63F1 Mice (gavage studies). NTP Tech. Rep. Ser. 289.

Nunes and Benville. 1979. As cited in Ballou et al., 1985.

Odum, W.E., G.M. Woodwell, and C.F. Wurster. 1969. DDT residues absorbed from organic detritus by fiddler crabs. *Science* 164:576-577.

Ohlendorf, H.M., R.W. Risebrough, and K. Vermeer. 1978. Exposure of Marine Birds to Environmental Pollutants. U.S. Fish and Wildlife Service, Wildlife Research Report 9, Washington, D.C.

Orberg and Lundberg. 1974. As cited ATSDR, 1989c.

Pasha, S. 1981. As cited in ATSDR, 1989c.

Pavlou, S.P. 1987. The Use of Equilibrium Partitioning Approach in Determining Safe Levels of Contaminants in Marine Sediments. Pages 388-412 in *Fate and Effects of Sediment-Bound Chemicals*, edited by K.L. Dickson et al. New York: Pergamon Press.

Perwak, J. et al. 1980. As cited in BEIA, 1989.

POLA. 1983. Consolidated Slip Dredging Project, including letter dated March 30, 1983, from E.L. Gorman (Chief Harbor Engineer) to Colonel P.W. Taylor (USACE

District Engineer), subject: Dredging in Consolidated Slip—Application No. 82-189-HB (SAC ACE 024 0016).

Porter, R.D., and S.N. Wiemeyer. 1972. DDE at low dietary levels kills captive American kestrels. Bull. Environ. Contam. Toxicol. 8:193-199.

Powers, C.D., C.F. Wurster, and R.G. Rowland. 1975. The toxicity of DDE to a marine dinoflagellate. Environ. Pollut. 9:253-262.

Powers, C.D., C.F. Wurster, and R.G. Rowland. 1979. Pestic. Biochem. Physiol. 10:306-312.

Rao et al. 1980. As cited in ATSDR, 1989d.

Rao, K.S. et al. 1980. Teratogenicity and reproduction studies in animals inhaling ethylene dichloride. Banbury Report 5:149-166.

Reddy, M.S., and K.V.R. Rao. 1986. Acute toxicity of insecticides to penaeid prawns. Environ. Ecol. 14:221-223.

Reddy, M.S., and K.V.R. Rao. 1989. Effects of phosophamidon and lindane on the limb regeneration of penaeid prawn, *Penaeus monodon*. Bull. Environ. Contam. Toxicol. 42:154-158.

Reish, D.J. 1959. An ecological study of pollution in Los Angeles-Long Beach Harbors, California. Allan Hancock Found. Occas. Paper 22.

Reish, D.J. et al. 1978. J. Water Pollut. Control Fed. 50:1424-1469. As cited in HSDB, 1992.

Remsen, J.V., Jr. 1978. Bird Species of Special Concern in California: an Annotated List of Declining or Vulnerable Bird Species. Wildlife Administrative Branch Administrative Report No. 78. CDFG. Sacramento.

Richter, M. 1992. Personal Communication between Mark Richter/Port of Los Angeles and Susan Keydel/CH2M HILL. July 1.

Risebrough et al. 1971. Reproductive failure of the brown pelican on Anacapa Island in 1969. Am. Birds 25:8-9.

Risebrough, R.W., and D.W. Anderson. 1975. Some effects of DDE and PCB on mallards and their eggs. J. Wildl. Manage. 39:508-513.

RTECS Data Base. 1989. As cited in HSDB, 1992.

\_\_\_\_\_. 1984. Available through the National Library of Medicine's MEDLARS System, NIOSH.

Rudolf, S.G. et al. 1984. Prey-capturing ability of American kestrel fed DDE and acephate or acephate alone. *Arch. Environ. Contam. Toxicol.* 13:367-372

Sabrurin and Tullis. 1981. As cited in Ballou et al., 1985.

SCAQMD. 1991.

Schimmel, S. C., J. M. Patrick, and J. Forester. 1977. Toxicity and bioconcentration of BHC and lindane in selected estuarine animals. *Arch. Environ. Contam. Toxicol.* 6:355-363.

Schwetz, B.A., B.K.J. Leong, and P.J. Gehring. 1974. Embryo- and fetotoxicity of inhaled chloroform in rats. *Toxicol. Appl. Pharmacol.* 28:442-451.

Seidenberg, J.M., D.G. Anderson, and R.A. Becker. 1986. Validation of an in vivo developmental toxicity screen in the mouse. *Teratog. Carcinog. Mutagen.* 6:361-374.

Shivanandappa and Krishnakumari, 1983. As cited in ATSDR, 1989f.

Sleicher, C., and J. Hopcraft. 1984. Persistence of pesticides in surface soil and relation to sublimation. *Environ. Sci. Technol.* 18:514-518.

Smith, S. I. et al. 1969. The effect of high levels of dietary DDT on egg production, mortality, fertility, hatchability and pesticides content of yolks in Japanese quail. *Poultry Sci.* 48:1000-1004.

Smokler, P. E., D.R. Young, and K.L. Gard. 1979. DDTs in marine fishes following termination of dominant California input: 1970-77. *Mar. Pollut. Bull.* 10:331-334.

Soule, D.F., and M. Oguri (eds). 1980. The Marine Environment in Los Angeles and Long Beach Harbors During 1978: Marine Studies of San Pedro Bay, California, Part 17. Harbors Environmental Projects, University of Southern California, Los Angeles, California. June.

Spencer, H.C. et al. 1951. Vapor toxicity of ethylene dichloride determined by experiments on laboratory animals. *Arch. Ind. Hyg. Occup. Med.* 4:482-493.

Stickel, L.F. 1973. Pesticide residues in birds and mammals. Pages 254-312 in *Environmental Pollution by Pesticides*, edited by C.A. Edwards. London and New York: Plenum Press.

SWRCB. 1992. Amendments of the Water Quality Control Plan for Enclosed Bays and Estuaries of California, Draft. Functional Equivalent Document. March 27.

Tatum et al. 1978. As cited in Ballou et al., 1985.

TDB Data Base. 1984. Available through the National Library of Medicine's MEDLARS System, National Library of Medicine TDB Peer Review Committee.

TDB Data Base. 1984. As cited in BEIA, 1989.

Tomatis, L. et al. 1974. Effect of long-term exposure to 1,1-dichloro-2,2-bis (p-chlorophenyl) ethylene to 1,1-dichloro-2,2-bis (p-chlorophenyl) ethane, and to the two chemicals combined on CF-1 mice. J. N. C. I. 52:883-891.

Torkelson, T.R., F. Oyen, and V.K. Rowe. 1976. The toxicity of chloroform as determined by single and repeated exposure of laboratory animals. J. Am. Ind. Hyg. Assoc. 37:697-705.

Truesdale Laboratories, Inc. 1972. As cited in CEC, 1986.

Tucker, R.K., and M.A. Haegele. 1970. Eggshell thinning as influenced by method of DDT exposure. Bull. Environ. Contam. Toxicol. 5:191-194.

Ungvary, G. 1985. The possible contribution of industrial chemicals (organic solvents) to the incidence of congenital defects by teratogenic drugs and consumer goods: An experimental study. Prog. Clin. Biol. Res. 163B: 295-300.

Ungvary, G. et al. 1980. Studies on the embryotoxic effect of ortho-meta-, and para-xylene. Toxicology 18:61-74.

Ungvary, G., and E. Tatrai. 1985. On the embryotoxic effects of benzene and its alkyl derivatives in mice, rats, and rabbits. Arch. Toxicol. Suppl. 8:425-430.

Ungvary and Tatrai. 1985. As cited in ATSDR, 1990c, 1989e.

Vermeer, K., and L.M. Reynolds. 1970. Organochlorine residues in aquatic birds in the Canadian prairie provinces. Can. Field-Nat. 84:117-130.

Vermeer, K., and R.W. Risebrough. 1972. Additional information on egg shell thickness in relation to DDE concentrations in great blue heron eggs. Can. Field-Nat. 86:384-385.

Vernberg et al. 1977. As cited in Ballou et al., 1985.

Verschueren, K. 1983. *Handbook of Environmental Data on Organic Chemicals*. Second Edition. New York: Van Nostrand Reinhold Co.

Verschueren, K. 1983. As cited in BEIA, 1989.

Vozovaya, M.A. 1974. Development of offspring of two generations obtained from females subjected to the action of dichloroethane. *Gig. Sanit.* 7:25-28.

Whitehead, C.C., J.N. Downie, and J.A. Phillips. 1974. Some characteristics of the egg shells of quail fed gamma-BHC. *Pestic. Sci.* 5:275-279.

Wiemeyer, S.N. et al. 1986. DDE, DDT, and Dieldrin: Residues in American Kestrels and Relations to Reproduction. United States Fish and Wildlife Service. Technical Report 6.

Wolf, M.A. et al. 1956. Toxicological studies of certain alkylated benzenes and benzene. *Arch. Ind. Health.* 14:387.

Wolfe, J. L., and R. J. Esher. 1980. Toxicity of carbofuran and lindane to the old field mouse (*Peromyscus polionotus*) and the cotton mouse (*P. gossypinus*). *Bull. Environ. Contam. Toxicol.* 24:894-902.

WHO. 1979. Environmental Health Criteria 9. DDT and its Derivatives. Geneva: World Health Organization.

\_\_\_\_\_. 1985. Toluene. Environmental Health Criteria 52. Geneva: World Health Organization.

\_\_\_\_\_. 1979. As cited in HSDB, 1992.

Young, D.R., D.J. McDermott, and T.C. Heesen. 1976a. DDT in sediments and organisms around southern California outfalls. *J. Water Pollut. Control Fed.* 48:1919-1928.

Young, D.R., D.J. McDermott, and T.C. Heesen. 1976b. Aerial fallout of DDT in southern California. *Bull. Environ. Contam. Toxicol.* 16:604-611.

Young, D.R. and T.C. Heesen. 1974. Inputs and distributions of chlorinated hydrocarbons in three Southern California harbors. TM 214. El Segundo, CA: Southern California Coastal Water Research Project.

Young, D.R., A.J. Mearns, and R.W. Gossett. 1991. Bioaccumulation of p,p'-DDE and PCB 1254 by a flatfish bioindicator from highly contaminated marine sediments of southern California. In: R.A. Baker, (ed) Organic substances in sediment and water. Volume 3, Biological. Chelsea, Michigan: Lewis Publishers.

Young, D.R., T.C. Heesen, and D.J. McDermott. 1980. Synoptic Survey of Chlorinated Hydrocarbon Inputs to the Southern California Bight. Volumes I and II (Revised). Southern California Coastal Water Research Project Authority, Long Beach, CA. June.

Zaroogian, G.E. et al. 1985. As cited in HSDB, 1992.

### References Reviewed But Not Cited

Many of these references, reviewed but not cited, were made available from the Department of Justice (DOJ). For those documents, the DOJ file number is provided.

Anderson, G.A. U.S. Congress. Letter to Paul R. Bonderson, Chief, Water Quality Division. January 14, 1970 (DOJ, MWB002, 1689).

Baldwin, Marine Editor. Dominguez Channel Waste Discharge Cut Fish Return (After waste discharge to Dominguez was cut, water quality improved and fish returned to channel). October 5, 1970 (DOJ, MWB002, 1646-1649).

Bearse, J.L., Starkist. No Title. 1987 (DOJ NFR008, 0089-0095).

Boyarsky, Bill. Los Angeles Harbor Port on Stormy Sea of Infighting, Political Squabbling Could Hurt Prospects for Future Growth, Ports Facing Many Challenges. October 29, 1973 (DOJ, SLC002, 0999-1000).

California Regional Water Quality Control Board, L.A. Region. Wilmington Drain-Harbor Lake Monitoring & Management Committee—Second Monitoring Data Report August 1974 Through September 1977. November 1, 1977.

Cloyd, L.H. Memo to Raymond Hertel, LARWQCB. January 24, 1968 (DOJ, MWB002, 1950).

Cloyd, Lawrence, California Dept. of Fish and Game. Recommendations of the Department of Fish and Game for Reconsideration of Beneficial Uses of Waters of Los Angeles Inner Harbor and Dominguez Channel. April 10, 1969 (DOJ, SFG001, 1215-1222).

Connell, Rich, Times Staff Writer. Fishing For Food Increases But So Does Toxic Danger. April 10, 1983.

Court Testimony. No Date (DOJ, SFG001, 0598-0615).

California Department of Water Resources. Tentative Requirements for 18 Firms Discharging Industrial Wastes to Dominguez Channel. February 20, 1968.

Department of Justice (DOJ). 1970. Report on Fishing Potential; Dominguez Flood Control Channel From Vermont Avenue to Los Angeles Harbor.

\_\_\_\_\_. 1982. Presentation to the U.S. Army Corps of Engineers, Los Angeles District-Los Angeles Harbor Department Consolidated Slip Dredging Configurations. September 14 (DOJ, MEP003, 1282-1285).

\_\_\_\_\_. Figure 3 and Table 8. No Date. pp. 23-24 (DOJ, SFG001, 1618-1619).

\_\_\_\_\_. No Title. 1983-1984 (DOJ, ACE024, 1940-1949).

\_\_\_\_\_. No Title. No Date (DOJ, NFR008, 0010).

\_\_\_\_\_. No Title. No Date (DOJ, MWB001, 1185-1187).

\_\_\_\_\_. No Title. No Date (DOJ, MWB001, 1221-1223).

Donkle, Royce. Dominguez Channel Users Group. May 12, 1967 (DOJ, SFG001, 1433-1434).

E., M. Dominguez Channel Dump Sites File 56122 (Reply to Memo: Available information given). February 18, 1957 (DOJ, MWB002, 1420-1430).

Envirologic Data, Inc. 1990. Selection of Indicator Chemicals for the Montrose Chemical Corporation of California Site, Torrance, California. November 19.

Fox, W.J. Dominguez Channel Sampling Points. February, 1954 (DOJ, SFG009, 1184-1186).

Fraser, J.C., Water Projects Branch. WP Los Angeles Inner Harbor and Dominguez Slough (Will submit history of Dominguez Channel and Inner Harbor and description of oil spills). September 19, 1967 (DOJ, SFG001, 1082).

Frost, Ernest, Calif. Dept. of Fish and Game. Funds for Bioassays of Outfalls Into Dominguez Channel. May 5, 1961.

G., Robert D. Proposed Action Regarding Dominguez Channel "Clean Up" Operation. February 3, 1961.

Gorman, E.L., Port of Los Angeles. Letter to Paul W. Taylor, District Engineer. March 30, 1983, (DOJ, ACE024, 0036-0038).

H+A. Field Data Submittal Part 2 Remedial Investigative Work Phase 2A Montrose Site Volume I 880889 To 880999 Dominguez Channel Sediment Survey and Sampling. December 2, 1988.

\_\_\_\_\_. Letter to Ms. Johanna Miller. November 22, 1989.

\_\_\_\_\_. 1989. Work Plan for the Public Health and Environmental Evaluation for the Montrose Site, Torrance, California. September 8.

Hertel, R.M. Letter to L.A. County Flood Control District. November 17, 1970 (DOJ, MW8002, 1645).

Hertel, R.M., LARWQCB. Application of Bays and Estuaries Policy to Dominguez Channel (Discussion of Policy Offers, Personal Thoughts and Recommendations). February 7, 1975.

Higbee, R.C., Corps of Engineers. Public Notice, Review of Section 404 Jurisdiction, Madrona Marsh, Torrance, CA. March 26, 1982 (DOJ, NFR023, 0822-0823).

Hurst, C. and T. Bradley, City of Los Angeles. L.A. Harbor Deepening Project Impacts to Marine Biota and Habitats. April 4, 1980.

Hurst, W.C., Port of Los Angeles. Letter to Paul W. Taylor, District Engineer. November 5, 1982 (DOJ, ACE024, 0142-0144).

ICF Technology, Inc. Review of Analytical Data. August 21, 1991.

ICF Technology, Inc. Review of Analytical Data. November 6, 1991.

Janssen, J.F., Calif. Dept. of Fish and Game. Pollution in Dominguez Channel. March 13, 1961.

Jones, R.A., Los Angeles Times. Breeding Ground for Rare Bird Becomes Parking Lot. April 27, 1978 (DOJ, IFW041, 1463).

Larson, L.C., Regional Water Pollution Control Board Region 4. Inter-Departmental Communication to Remo Navone, Branch Public Health Laboratory. March 11, 1965 (DOJ MWB002, 2030).

Long Beach Harbor Department. Draft Environmental Impact Statement: Environmental Impact Report Phase I: 2020 Plan and Feasibility Study (Channel Improvements and Landfill Development). September, 1990. pp. 1.1-10.21 (DOJ, ACE009, 1608-2440).

L.A. Department of County Engineer, Flood Control District. L.A. County Sanitation Districts, Disposal of Wastes into the Pacific Ocean Los Angeles Long Beach Harbor Area and Dominguez Channel. 1967.

Los Angeles Department of County Engineer, Flood Control District. L.A. County Sanitation Districts, Disposal of Wastes into the Pacific Ocean Los Angeles Long Beach Harbor Area and Dominguez Channel. 1967. pp. 4-26.

Los Angeles Harbor. Consolidated Slip Dredging Project. 1982 (DOJ, ACE024, 0016).

Los Angeles Harbor Department. Phase I:2020 Plan and Feasibility Study, Channel Improvements and Landfill Development, Draft Environmental Impact Statement/Environmental Impact Report. July, 1990. Vol. 1:1.1-10.22 (DOJ, ACE001, 0934-1419).

Los Angeles Harbor Dept. Los Angeles Harbor Department Consolidated Slip Dredging Configurations. September 14, 1982 (DOJ, ACE024, 0010-0013).

Los Angeles Herald. Firms Plan 2.89 Million Expense for Prevention. March 19, 1968 (DOJ, MWB002, 1926-1927).

Los Angeles Regional Water Quality Control Board. Review of Information Pertinent to Los Angeles-Long Beach Harbor and Dominguez Channel; A Staff Report to the LARWQCB. May, 1969. pp. I.1-VIII.10 (DOJ, MEP027, 1097-1187).

MacGregor, J.S. 1976. DDT and Its Metabolites in the Sediments off Southern California. Fishery Bulletin Vol. 74, No. 1, pp. 27-35.

Marine Bioassay Laboratories. Technical Evaluation of Environmental Impact Potential for Proposed Ocean Disposal of Dredged Material from Los Angeles Harbor, Final Report, Material Proposed for Dredging from Berths 200G and H Dominguez Channel. November, 1981. pp. 1-41 (DOJ, NFS022, 2016-2061).

Marine Bioassay Laboratories. Technical Evaluation of Environmental Impact Potential for Proposed Ocean Disposal of Dredged Material from Los Angeles Harbor (statistical results only). No Date. pp. 7-9 (DOJ, ACE024, 0311-0313).

May, A.E. Memo to Dominguez Channel Users. July 7, 1970 (DOJ, MWB002, 1657).

McCanna. Maintenance Dredging Project Location Maps. January, 1987 (DOJ, SLC003, 1127-1130).

McCauley, J. Mailing List for Special Meeting—Water Quality Control Policy For Harbors, Marinas, and Tidal Prisms of Los Angeles and Ventura Counties. April, 1967 (DOJ, MWB005, 2342).

Mckee, J.E. Temperature Studies (For Dominguez Channel and Detailed Data for each Discharger. March 19, 1971.

Meyerson, L.A., LARWQCB. Letter to L.A. City Bureau of Sanitation and L.A. County Flood Control District. May 2, 1969 (DOJ, MWB002, 1720).

Montgomery James M., Consulting Engineers, Inc. No Title. No Date. pp. 1-10 (DOJ, MWB002, 2035-2046).

Montgomery, R.D. 1960. Long Beach Water Quality Objectives and a Plan for the Improvement of Water Quality Conditions in Los Angeles Harbor and Dominguez Channel. September 18. pp. 1-6 (DOJ, SFG001, 1209-1214).

National Oceanic and Atmospheric Administration (NOAA). 1991. Contaminant Trends in the Southern California Bight: Inventory and Assessment. NOAA Technical Memorandum NOS ORCA 62 Seattle, WA. October.

Patez, L. Work Order. June 15, 1973 (DOJ, SLC003, 0314-0315).

Port of Los Angeles. Proposed Dredging in Consolidated Slip. July, 1982 (DOJ, MEP003, 1058).

Prescott, J.H. Los Angeles Harbor Bio-Assay Investigations Union Oil Wilmington California Dominguez Channel Discharge, Wilmington, California. No Date. pp. 1-25. Duplicate of 65 (DOJ, SFG009, 0835-0859).

Prescott, J.H. Los Angeles Harbor Bioassay Investigations (Union Oil, Wilmington, California Dominguez Channel Discharge). July 29, 1958. pp. 1-25. Duplicate of 64 (DOJ, SFG001, 0951-0976).

Regional Supervisor, Office of Leasing and Environment USDOJ. Memo to Regional Supervisor, Office of Field Operations, USDOJ. March 6, 1991.

Rice, D. City of Los Angeles. L.A. Harbor Deepening Project Impacts To Marine Biota and Habitats. April 4, 1980.

Schinazi, L.A. CRWQCB, L.A. Region. Urban Lakes Health Effects Study/Preliminary Report. December 14, 1983.

Steiger, F., Port of Los Angeles. Memo to E.L. Gorman, Chief Harbor Engineer. April 28, 1981 (DOJ, NFS022, 1980).

Streeter, G.N. Letter to L.A. County Engineer. November 17, 1970 (DOJ, MWB002, 1644).

Wood, W.J., Los Angeles County Flood Control District. Tentative Requirements for Waste Discharges Into Dominguez Channel Tidal Prism. February 16, 1968.

Workman, J.M. Letter to R.A. Harris. April 20, 1971 (DOJ, MWB002, 1634-1635).

Yaghoubian, N.F., Dames and Moore. Predredging Investigation Proposed LNG Ship Channel Los Angeles Harbor For Pacific AK LNG Company. No Date. pp. 15-16 (DOJ, ACE001, 0020-0021).

# Appendix A

## Bioconcentration Factors for Aquatic Organisms

Appendix A Bioconcentration Factors for Aquatic Organisms			
Chemical	Species	BCF	Reference
DDT	Snails ( <i>Cipangopaludina japonica</i> )	3,660 [1]-34,500 [2]	[1] Verschuereen, 1983. [2] Metcalf et al., 1973.
	Mussels ( <i>Mytilus edulis</i> )	4,550 [3]-690,000 [4]	[3] Geyer et al., 1982. [4] Reish et al., 1978.
	Oysters ( <i>Crassostrea virginica</i> )	700 - 70,000	Verschuereen, 1983.
	Coontail ( <i>Ceratophyllum demersum</i> ) [30-day exposure]	1,950	Eberhardt et al., 1971.
	<i>Cladophora</i> sp. [30-day exposure]	21,580	Eberhardt et al., 1971
	Duckweed ( <i>Lemna minor</i> ) [30-day exposure]	1,210	Eberhardt et al., 1971.
	Water milfoil ( <i>Myriophyllum</i> ) [30-day exposure]	1,870	Eberhardt et al., 1971.
	Curly leaf pondweed ( <i>Potamogeton crispus</i> ) [30-day exposure]	14,280	Eberhardt et al., 1971.
	Narrow leaf pondweed ( <i>Potamogeton follo-</i> <i>sus</i> ) [30-day exposure]	781	Eberhardt et al., 1971.
	Sago pondweed ( <i>Potamogeton pectinatus</i> ) [30-day exposure]	6,360	Eberhardt et al., 1971.
	Bur reed ( <i>Sparganium eurycarpum</i> ) [30-day exposure]	623	Eberhardt et al., 1971.
	Bladderwort ( <i>Utricularia vulgaris</i> ) [30-day exposure]	2,200	Eberhardt et al., 1971.
	Crayfish ( <i>Orconectes punctata</i> ) [30-day exposure]	5,060	Eberhardt et al., 1971.
	Bloodworm ( <i>Tendipes</i> sp.) [30-day exposure]	4,750	Eberhardt et al., 1971.
	Red Leach ( <i>Eryopodella punctata</i> ) [30-day exposure]	7,520	Eberhardt et al., 1971.
	Green Algae ( <i>Ulva rigida</i> )	7	Andryushchenko and Polikarpov, 1974.
	Green Algae ( <i>Enteromorpha chuii</i> )	0.08	Sikka and Rice, 1972.
	Haptophyte ( <i>Isochrysis galbana</i> )	0.08	Sikka and Rice, 1972.
	Diatom ( <i>Skeletonema costatum</i> )	0.08	Sikka and Rice, 1972.
	Diatom ( <i>Thalassiosira guillardii</i> )	0.08	Sikka and Rice, 1972.
	Dinoflagellate ( <i>Amphidinium carterae</i> )	0.08	Sikka and Rice, 1972.
	Dinoflagellate ( <i>Olisthodiscus luteus</i> )	0.08	Sikka and Rice, 1972.
DDE	Snail ( <i>Cipangopaludina japonica</i> ) [terres- trial-aquatic microcosm, 3.8 µg/L in water]	36,000	Callahan, 1979.
	Mosquito larvae [terrestrial-aquatic micro- cosm, 3.8 µg/L in water]	59,000	Callahan, 1979.
	Snail ( <i>Cipangopaludina japonica</i> ) [aquatic model ecosystem]	13,700	Verschuereen, 1983.
	Alga [aquatic model ecosystem]	2,720	Verschuereen, 1983.
DDD	Alga	6,210	Verschuereen, 1983.
	Snail ( <i>Cipangopaludina japonica</i> )	4,460	Verschuereen, 1983.
	Pelecypod (0.05-2.18 µg/L/50hr)	9,210	Verschuereen, 1983.
	Mussel ( <i>Mytilus edulis</i> )	9,120	Zarogian et al., 1985.
	Oyster ( <i>Crassostrea virginica</i> )	47,900	Zarogian et al., 1985.

Continued

Appendix A Bioconcentration Factors for Aquatic Organisms			
Chemical	Species	BCF	Reference
Technical BHC <sup>a</sup>	American oyster, all soft tissue ( <i>Crassostrea virginica</i> ) [28-day exposure]	218	Veith and Kosian, 1983.
	Pinfish, edible tissue ( <i>Lagodon rhomboides</i> ) [28-day exposure]	130	Veith and Kosian, 1983.
	Pinfish, offal tissue ( <i>Lagodon rhomboides</i> ) [28-day exposure]	617	Veith and Kosian, 1983.
	Pink shrimp ( <i>Penaeus duorarum</i> ) [4-day exposure]	80	Veith and Kosian, 1983.
	Pinfish, ( <i>Lagodon rhomboides</i> ) [4-day exposure]	482	Veith and Kosian, 1983.
Lindane	Grass shrimp ( <i>Palaemonetes pugio</i> ) [4-day exposure]	63	Veith and Kosian, 1983.
	Pink shrimp ( <i>Penaeus duorarum</i> ) [4-day exposure]	84	Veith and Kosian, 1983.
	Sheepshead minnow ( <i>Cyprinodon variegatus</i> ) [4-day exposure]	490	Veith and Kosian, 1983.
	Pinfish ( <i>Lagodon rhomboides</i> ) [4-day exposure]	218	Veith and Kosian, 1983.
<sup>a</sup> Technical BHC (21 percent alpha BHC, 2.1 percent beta BHC, 39 percent gamma BHC, 23 percent delta BHC, 14.9 percent unidentified compounds) Source: TOXNET, Hazardous Substances Data Bank ASTER Ecotoxicity Profile, USEPA Environmental Research Laboratory, Duluth.			

Other chemicals considered in this risk assessment have little or no tendency to bio-concentrate in aquatic communities, as summarized below:

### Benzene

BCF for eels is 3.1 [1], and for goldfish is 4.3 [3]. Based on reported BCF of 2.13 [4] and an estimated BCF of 24 [5], benzene will not be expected to bio-concentrate in aquatic organisms.

[1] Ogata, M., and Y. Miyake. 1978. Water Res. 12:1041-4.

[3] Ogata, M. et al. 1984. Bull. Environ. Contam. Toxicol. 33:561-7.

[4] Hansch, C., and A.J. Leo. 1985. Medchem Project Issue No. 26. Claremont, CA: Pomona College.

[5] Lyman, W.J. et al. 1982. *Handbook of Chemical Property Estimated Methods*. NY: McGraw-Hill.

## Chlorobenzene

Based on a log BCF of 1-2 for several species of fish [1,3,4], it is concluded that chlorobenzene has little or no tendency to bioconcentrate. [1,2,3,4].

- [1] Kenaga, E.E. 1980. Bull. Environmental Safety. 4:26-38.
- [2] Veith, G.D. et al. 1979. Journal Fish. Board Canada. 36:1040-48.
- [3] Kawasaki, M. 1980. Ecotoxicol. Environ. Safety. 4:444-54.
- [4] Kitano, M. 1978. OECD Tokyo Mtg. Ref. Book TSU-No. 3.

## Chloroform

Based on the log BCF < 1 for 4 species of fish, it is concluded that chloroform has little or no tendency to bioconcentrate [1,2].

- [1] Barrows, B.E. et al. 1980. *Dynamic Exposure Hazard Assessment Toxic Chemical*. Ann Arbor, MI: Ann Arbor Press. pp. 379-92.
- [2] Anderson, D.R., and E.B. Lusty. 1980. Acute Toxicity and Bioaccumulation of Chloroform to Four Species of Fresh Water Fish, NUREG/CR-089, Richland, WA, Pacific NW Labs. pp. 8-26.

## 1,2-DCA

1,2-Dichloroethane is not expected to bioconcentrate in fish because of its low octanol/water partition of 1.48. [1]. The measured log BCF in bluegill sunfish is 0.30 [2].

- [1] Hansch, C., and A.J. Leo. 1979. *Substituent Constants for Correlation Analysis in Chemistry and Biology*, New York, NY: John Wiley and Sons.
- [2] Barrows, M.E. et al. 1980. *Dynamic Exposure Hazard Assess. Toxic Chem.* Ann Arbor, MI: Ann Arbor Sci. pp. 379-92.

## Ethylbenzene

The only experimental data on the bioconcentration of ethylbenzene are the low log BCF of 0.67 for clams exposed to the water-soluble fraction of crude oil [1]. The bioconcentration factor can be calculated for fish based on the log octanol/water partition coefficient of 2.16 [2] and a recommended regression equation [3]. The calculated BCF for fish is 2.16. It is concluded that ethylbenzene should not significantly bioconcentrate in aquatic organisms.

[1] Nunes, P., and P.E. Benville, Jr. 1979. Bull. Environ. Contam. Toxicol. 21:719-24.

[2] Hansch, C., and A.J. Leo. 1981. Medchem Project Issue No. 19. Claremont, CA: Pomona College.

[3] Lyman, W.J. et al. 1982. *Handbook of Chemical Property Estimation Methods*. Environmental Behavior of Organic Compounds. New York, NY: McGraw Hill. pp. 5-1 to 5-10.

### **Toluene**

Based on the log BCF range of 0.22 to 1.12 [1-4] for fish and aquatic invertebrates, it is concluded that toluene does not bioconcentrate significantly in fish and aquatic invertebrates.

[1] Ogata, M., and Y. Miyake. 1978. Water Res. 12:1041-4.

[2] Nunes, P. and P.E. Benville Jr. 1979. Bull. Environ. Contam. Toxicol. 12:719-24.

[3] Geyer, H. et al. 1982. Chemosphere 11:1121-34.

[4] Korn, S. et al. 1977. Fish. Bull. U.S. Dept. Commerce, NOAA Natl. Mar. Fish Serv. 75:633-6.

### **Xylenes**

The bioconcentration factor can be calculated for fish based on the log octanol/water partition coefficient of 3.12-3.20 for the individual isomers [1] and a regression relation [2]. The calculated BCF for fish is 2.14-2.20. The calculated BCF for eels is 1.3 [3].

[1] Hansch, C., and A.J. Leo. Medchem Project No. 19. Claremont, CA: Pomona College.

[2] Lyman, W.J. et al. 1982. *Handbook of Chemical Property Estimation Methods*. New York, NY: McGraw Hill, p. 5-5.

[3] Ogata, M., and Y. Miyaka. 1978. Water Res. 12:1041-4.

## References for Appendix A

### Bioconcentration Factors for Aquatic Organisms

Andryushchenko, V.V. and G.G. Polikarpov. 1974. An experimental study of uptake of Zn<sup>65</sup> and DDT by *Ulva rigida* from seawater polluted with both agents. *Hydrobiol. J.* (Engl. Transl. *Gidrobiol. Zh.*). 10 (4):41-46. As cited in ASTER Ecotoxicity Profile, USEPA Environmental Research Laboratory, Duluth.

Callahan. 1979. Water-related environmental fate of priority pollutants. 1:24-7. As cited in TOXNET, Hazardous Substances Data Bank.

Eberhardt, L.L. et al. 1971. *Nature* 230:60. As cited in USEPA Ambient Water Quality Criteria Doc: DDT. p. B-36 - B-37. 1980. Report No. EPA 440/5-80-038. As cited in TOXNET, Hazardous Substances Data Bank.

Geyer, H. et al. 1982. *Chemosphere* 11. pp. 1121-34. As cited in TOXNET, Hazardous Substances Data Bank.

Metcalf, R.L. et al. 1973. Model ecosystem studies of the environmental fate of six organochlorine pesticides. *Environ. Health Perspect.* 4:35-44. As cited in ASTER Ecotoxicity Profile, USEPA Environmental Research Laboratory, Duluth.

Reish, D.J. et al. 1978. *J. Water Pollut. Control Fed.* 50:1424-1469. As cited in TOXNET, Hazardous Substances Data Bank.

Sikka, H.C. and C.P. Rice. 1972. Uptake and Metabolism and DDT and Dieldrin by Marine Algae. Annual Report. No. 1. Syracuse University. Res. Corp. Life Science Division. U.S. NTIS AD-744 034. p. 36. Government Reports Announcements and Index 7215. As cited in ASTER Ecotoxicity Profile, USEPA Environmental Research Laboratory, Duluth.

Veith, G.D. and P. Kosian. 1983. Estimating bioconcentration potential from octanol/water partition coefficient as cited in D. Mackay et.al. *Physical Behavior of PCBs in the Great Lakes*. Ann Arbor, Michigan: Ann Arbor Science Publishers. 269-282 pp. As cited in ASTER Ecotoxicity Profile, USEPA Environmental Research Laboratory, Duluth.

Verscheuren, K. 1983. *Handbook of Environmental Data on Organic Chemicals*. Second Edition. New York: Van Nostrand Reinhold Co. As cited in TOXNET, Hazardous Substances Data Bank.

Zaroogian, G.E. et al. 1985. *Environ. Toxicol. Chem.* 4:3-12. As cited in TOXNET, Hazardous Substances Data Bank.

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
DDT (Matsumura, 1985)	American kestrels			1.4 & 4.7 mg/kg, diet	Birds produced eggshells roughly 10% thinner than normal ones.		
DDT (Amdur et al., 1980)	Rats	<200 mg/kg, diet	No adverse effects in female rats only.	5-15 mg/kg, diet	6-month exposure produced histologic changes in livers of male rats including hypertrophy, inclusion bodies, and cytoplasmic granulation in male rats only. Female rats developed liver necrosis at greater than 1,000 mg/kg in diet.		
DDT (Clarke, 1981)	Domestic fowl			0.1-0.3%, diet	Sperm production greatly reduced; signs of toxicity followed.		
DDT (Cabral, 1982)	MRC Proton rats	500 mg/kg, diet	Lifetime exposure produced no adverse effects on body growth or survival rate. No increase in incidence of liver-cell tumors in females.				
DDT (Shepard, 1986)	Rabbits			50 mg/kg body weight, diet	Exposure on day 7, 8 & 9 of gestation produced premature delivery, increased fetal resorptions, and reduced intrauterine growth.		
Continued							

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
				1 mg/kg, diet	Exposure on days 4 through 7 of gestation resulted in reduction of brain weight in rabbit fetuses.		
DDT (Wurster, 1968)	Bermuda petrels					6.4 mg/kg	Dead chicks resulted from Bermuda petrels feeding on cephalopods containing DDT and DDE
DDT (Ballou et al., 1985)	Blue crabs ( <i>Callinectes sapidus</i> )			0.8 mg/L, food	Increased respiratory rates		
DDT (Smith et al., 1969)	Coturnix quail (5 weeks old)	100 mg/kg, diet	No difference in mortality, fertility, or hatchability from controls				
DDT (Hudson et al., 1984)	Mallard	30 mg/kg, diet	90 days exposure, no mortalities			100 mg/kg, diet	First mortality at 43 days, last at 417 days
	Bobwhite	30 mg/kg, diet	90 days exposure, no mortalities				
		100 mg/kg, diet	60 days exposure did not produce intoxication and eggshell thickness was normal				
	Sandhill crane					1,000 mg/kg/day, diet	Toxic signs by day 10; death after day 12
	Ring-necked pheasants, female, 3-4 months old					1,334 mg/kg	LD <sub>50</sub> , oral
	Sandhill crane, male and female					>1,200 mg/kg	LD <sub>50</sub> , oral
	Bullfrog, female					>2,000 mg/kg	LD <sub>50</sub> , oral
	Mallard, 3 months old					>2,240 mg/kg	LD <sub>50</sub> , oral

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
	Japanese quail, male, 2 months old					841 mg/kg	LD <sub>50</sub> , oral
	Rock dove (domestic pigeon); male and female					>4,000 mg/kg	LD <sub>50</sub> , oral
	California quail, male, 6 months old					595 mg/kg	LD <sub>50</sub> , oral
DDT (Fabro et al., 1984)	Rabbit (4-7 days gestation)			1 mg/kg/day, diet	Decreased fetal weight		
DDT (Hart et al., 1972)	Rabbit (7, 8, 9 or 21, 22, 23 days gestation)			10 mg/kg/day, diet	Increased fetal resorption		
DDT (Orberg and Lundberg, 1974)	Mouse	8.33 mg/kg/day, diet	28 days exposure did not produce reproductive effects				
DDT (Lowe et al., 1971)	Oyster	1 µg/L (submersion)	Weight and height not affected				
DDT (DeWaziers and Azias, 1987)	Rat	40 mg/kg/day, diet	12 days exposure did not produce gastrointestinal effects	40 mg/kg/day, diet	Effects on liver were apparent		
DDT (Pasha, 1991)	Mouse	26 mg/kg/day, diet	1-week exposure did not produce hepatic effects				
DDT (Gish and Chura, 1970)	Coturnix quail					700, 922, 1,214, 1,600 mg/kg, diet	Fed for 20 days or until death, mortality varied with body condition and sex.
DDT (Davison and Sell, 1974)	Mallard			20 mg/kg dry weight, diet	Significant eggshell thinning		
DDT (Jefferies, 1971)	Bengalese finches			8, 32, & 274 mg/kg, diet	Reduced fertility, hatchability, and fledging success.		
DDT (Worthing, 1987)	Rat					113-118 mg/kg	LD <sub>50</sub> , oral

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
DDT (Hartley, 1987)	Mouse					150-300 mg/kg	LD <sub>50</sub> , oral
	Rabbit					300 mg/kg	LD <sub>50</sub> , oral
	Dog					500-750 mg/kg	LD <sub>50</sub> , oral
	Sheep					>1,000 mg/kg	LD <sub>50</sub> , oral
	Goat					>1,000 mg/kg	LD <sub>50</sub> , oral
DDT (Hill et al., 1975)	Bobwhite quail, 23 days old					611 mg/kg	LC <sub>50</sub> , 5-day diet
	Japanese quail, 7 days old					568 mg/kg	LC <sub>50</sub> , 5-day diet
	Pheasant, 21 days old					311 mg/kg	LC <sub>50</sub> , 5-day diet
	Mallard, 17 days old					1,869 mg/kg	LC <sub>50</sub> , 5-day diet
DDT (Hill and Camardese, 1986)	Japanese quail, 2 weeks old					416 mg/kg	LC <sub>50</sub> , 5-day diet
DDT (Ferguson and Gilbert, 1967)	Toad					560,000 µg/L/36 hours	LC <sub>50</sub>
DDT (Sanders, 1970)	Toad, tadpole, 4-5 weeks old					1,000 µg/L/96 hours	LC <sub>50</sub>
	Toad, tadpole, 6 weeks old					100 µg/L/96 hours	LC <sub>50</sub>
	Toad, tadpole, 7 weeks old					30 µg/L/96 hours	LC <sub>50</sub>
	Frog, tadpole					400 µg/L/96 hours	LC <sub>50</sub>
DDT (EPA, 1980a)	Spot					1.8 µg/L/2 days	LC <sub>50</sub>
	Striped mullet					0.4 µg/L/2 days	LC <sub>50</sub>
DDT (McLeese and Metcalfe, 1980)	Sand shrimp ( <i>Crangon septemspinosa</i> )					0.63 µg/L	LC <sub>50</sub>
						0.83 µg/L	LC <sub>50</sub>

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
DDT (Meith-Avcin, 1974)	Barnacles ( <i>Balanus im-provises</i> )			Not given	Reduced settlement densities		
DDT (Odum et al., 1969)	Fiddler crabs ( <i>Uca pug-nax</i> )			10 mg/kg, diet	Uncoordination, slug-gish response after 5 days		
DDT (Ballou et al., 1985)	Fiddler crabs ( <i>Uca pug-nax</i> , <i>U. pugilator</i> )			10 mg/L, water	Increase in limb re-generation time		
p,p-DDE (IARC 1974)	CF-1 mice			250 mg/kg, diet	Lifetime exposure re-sulted in an increased incidence of liver tu-mors in male and female mice.		
p,p-DDE (Tomatis et al., 1974)	CF-1 mice			250 mg/kg, diet	130-week exposure re-sulted in a high inci-dence (100%) of liver tumors in female rats.		
p,p-DDE (Matsumura, 1985)	American kestrel			2.8 mg/kg, diet	Birds produced egg shells roughly 10% thinner than normal ones.		
DDE (Heinz, 1976)	Mallard			3 mg/kg, diet	Female mallards laid eggs containing an average of 5.8 mg/kg DDE. Ducklings were hyperresponsive to ma-ternal call and to frightening stimuli.		
DDE (Stickel et al., 1984)	4 species of wild birds					1,500 mg/kg, diet	Brain residues ranged from 305-694 mg/kg in dead birds, independent of bird species.
DDE (EPA, 1980a)	Saltwater aquatic life					14 µg/L	Lowest concentration to produce acute toxicity.

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
DDE (DHEW, 1978)	Osborne-Mendel rats					21.85 mg/kg/day, diet—male	Increased mortality was seen in both sexes of rats. No evidence of carcinogenicity was found in either sex. DDE was hepatotoxic and induced centrilobular necrosis and fatty metamorphosis.
						12.1 mg/kg/day, diet—female	
p,p'-DDE (Kornburst, 1986)	Female Sprague-Dawley rats	10 mg/kg body weight, diet	Exposure of 5 days/week for 5 weeks before mating through lactation periods did not adversely affect lactation or neonatal growth.				
DDE (Heath et al., 1969)	Mallard			10 & 40 mg/kg dry weight, diet	Eggshell thinning, cracking; increased embryo mortality		
DDE (Risebrough and Anderson, 1975)	Mallard			40 mg/kg dry weight, diet	17% average decrease in eggshell thickness		
				40 mg/kg + 40 mg/kg PCB, diet	19% average decrease in eggshell thickness		
DDE (Rudolf et al., 1984)	American kestrel	35 mg/kg DDE + 50 mg/kg acephate, diet	No appreciable effects on kestrels response to prey stimulus with which they have had extensive prior contact				
DDE (Jefferies, 1971)	Bengalese finches			4, 38, & 91 mg/kg, diet	Decrease fertility, hatchability, fledging success		
DDE (Miller et al., 1976)	Ducks	10-250 mg/kg, diet	No effects on nasal gland secretion or plasma osmores-regulation				
DDE (Powers et al., 1975)	Exuviella baltica (dino-flagellate)			0.1 parts per billion in cell culture <sup>b</sup>	Inhibition of cell growth		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
				10 parts per billion in cell culture <sup>b</sup>	Decrease in cell number		
DDE (Powers et al., 1979)	Exuviella baltica			25 µg/L in cell culture	Inhibition of cell growth, reduced photosynthesis		
DDE (Pasha, 1981)	Mouse			26 mg/kg/day, diet	24 hours/day exposure for 1 week resulted in liver damage		
DDE (Longcore et al., 1971)	Black duck			10 & 30 mg/kg dry weight, diet	Changes in eggshell composition significant eggshell thinning; increased eggshell cracking	46 mg/kg, diet	Reduced survival rate of 21-day duckling by 40-76%
	Mallard			5 & 10 mg/kg dry weight, diet	Changes in eggshell composition		
DDE (Ludke, 1974)	Coturnix quail			2 mg/kg DDE + 1 mg/kg dieldrin, diet	56 days exposure, DDE residues were significantly greater than in birds fed DDE alone		
DDE (Porter and Wiemeyer, 1972)	American kestrel					2.8 mg/kg, diet	14- to 16-month exposure, 2 of 14 males died; high brain residues of DDE
DDE (Longcore and Samson, 1973)	Black duck			10 mg/kg dry weight, diet	Thinning of eggshells, cracking		
DDE (Hill et al., 1975)	Bobwhite quail, 23 days old					825 mg/kg	LC <sub>50</sub> , 5-day diet
	Japanese quail, 7 days old					1,355 mg/kg	LC <sub>50</sub> , 5-day diet
	Ring-necked pheasant, 10 days old					829 mg/kg	LC <sub>50</sub> , 5-day diet
	Mallard					3,572 mg/kg	LC <sub>50</sub> , 5-day diet

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
DDE (Hill and Camardese, 1986)	Japanese quail					859 mg/kg	LC <sub>50</sub> , 5-day diet
DDD (Meister, 1989)	Rat					3,400 mg/kg	LC <sub>50</sub> , oral
DDD (Hill et al., 1975)	Bobwhite quail, 23 days old					2,178 mg/kg	LC <sub>50</sub> , 5-day diet
	Japanese quail, 7 days old					3,165 mg/kg	LC <sub>50</sub> , 5-day diet
	Ring-necked pheasant, 10 days old					445 mg/kg	LC <sub>50</sub> , 5-day diet
	Mallard, 17 days old					4,814 mg/kg	LC <sub>50</sub> , 5-day diet
DDD (Hill and Camardese, 1986)	Japanese quail					2,636 mg/kg	LC <sub>50</sub> , 5-day diet
DDD (Hudson et al., 1984)	California quail, female, 6 months old					>760 mg/kg	LD <sub>50</sub>
	Ring-necked pheasant, female, 3-4 months old					386 mg/kg	LD <sub>50</sub>
	Mallard, female, 3 months old					>2,000 mg/kg	LD <sub>50</sub>
DDD (Pasha, 1981)	Mouse	26 mg/kg/day, diet	1-week exposure, no hepatic effects				
DDD (Tomatis, 1974)	CF-1 mice			250 mg/kg, diet	Exposure of 130 weeks resulted in moderate increase of liver tumors in males only, and an increase of lung tumors in both sexes.		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
DDD (Clarke, 1981)	Dogs			20-50 mg/kg, diet	Daily exposure produces atrophy and disappearance of the deeper layers of the adrenal cortex. Effects are specific to o,p'-DDD, which may be a contaminant of p,p'-DDD.		
BHC (Fitzhugh et al., 1950)	Weanling Wistar rats	10, 50, 100, 800 mg/kg, diet	Lifetime exposure containing 64% alpha, 10% beta, 9% delta, 13% lindane isomers produced no increase incidence of tumors.				
BHC (Ito et al., 1973)	Male DD mice	250 mg/kg each beta + lindane, beta + delta, lindane+delta, diet	8-week-old male DD mice exposed for 24 weeks produced no liver tumors	250 mg/kg each alpha+delta, alpha+lindane, alpha+beta, diet	8-week-old mice exposed for 24 weeks. 25-50% treated developed liver tumors with no metastases.		
BHC (Hanada, 1973)	DD mice	100 mg/kg, diet	6-week-old males and females exposed for 32 weeks; no tumors were observed	300 mg/kg and 600 mg/kg, diet	6-week-old exposed for 32 weeks developed liver tumors. Average size of tumors was dose-related.		
BHC (Goto et al., 1972)	ICR-JCL mice			600 mg/kg, diet	5-week-old exposed for 26 weeks developed benign liver tumors		
BHC (Clarke, 1981)	Poultry					250-500 mg/kg, diet	Hemorrhages in intestinal mucous membranes, parenchymatous organs and other tissues.
BHC (Hudson et al., 1984)	Mallard, 3 months old					1,414 mg/kg	LD <sub>50</sub> , oral

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
	Pheasants, female, 3-4 months old					118 mg/kg	LD <sub>50</sub> , oral
BHC (EPA, 1980b) <sup>a</sup>	Pink shrimp ( <i>Penaeus duorarum</i> )					0.34 µg/L (mean for species—0.34 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; flow-through, measured
	Pinfish ( <i>Lagodon rhomboides</i> )					86.4 µg/L (mean for species—86.4 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; flow-through, measured
Lindane (Hill et al., 1975)	Bobwhite, 9 days old					882 mg/kg	LC <sub>50</sub> , 5-day diet
	Japanese quail, 7 days old					425 mg/kg	LC <sub>50</sub> , 5-day diet
	Pheasant, 10 days old					561 mg/kg	LC <sub>50</sub> , 5-day diet
	Mallard, 15 days old					5,000 mg/kg	LC <sub>50</sub> , 5-day diet
Lindane (Hill and Camardese, 1986)	Japanese quail					663 mg/kg	LC <sub>50</sub> , 5-day diet
Lindane (Hudson et al., 1984)	Mallard, 3-4 months old					>2,000 mg/kg	LD <sub>50</sub> , oral
Lindane (Schimmel et al., 1977)	Mysid shrimp					6.3 µg/L	LC <sub>50</sub> , 96 hours, flow-through
	Pink shrimp					0.17 µg/L	LC <sub>50</sub> , 96 hours, flow-through
	Grass shrimp ( <i>Palaemonetes pugio</i> )					4.4 µg/L	LC <sub>50</sub> , 96 hours, flow-through
	Sheepshead minnow					104 µg/L	LC <sub>50</sub> , 96 hours, flow-through
	Pinfish					30.6 µg/L	LC <sub>50</sub> , 96 hours, flow-through

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
Lindane (Whitehead et al., 1974)	Japanese quail	200 mg/kg, diet	No adverse effects observed on rate or pattern of egg production, egg size, shell thickness, calcium content, shearing strength, or structure.				
Lindane (Shivanandappa and Krishnakumari, 1983)	Rat	37.5 mg/kg/day, diet	90 days ad lib, no reproductive effects	75 mg/kg/day, diet	90 days ad lib, disrupted spermatogenesis		
Lindane (Reddy and Rao, 1986)	Penaeid prawn					0.1 mg/L	LC <sub>50</sub> , 96 hours
Lindane (EPA, 1980b)	Sand Shrimp ( <i>Crangon septemspinosa</i> )					5 µg/L (mean for species—5 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static, unmeasured
	Hermit Crab ( <i>Pagurus longicarpus</i> )					5 µg/L (mean for species—5 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static, unmeasured
	Sheepshead minnow ( <i>Cyprinodon variegatus</i> )					103.9 µg/L (mean for species—103.9 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; flow-through, measured
	Striped bass ( <i>Morone saxatilis</i> )					7.3 µg/L (mean for species—7.3 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; flow-through, unmeasured
	Pinfish ( <i>Lagodon rhomboides</i> )					30.6 µg/L (mean for species—30.6 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; flow-through, unmeasured
	Longnose killifish ( <i>Fundulus similis</i> )					240 µg/L	LC <sub>50</sub> , 48-hour exposure
	White mullet ( <i>Mugil curema</i> )					30 µg/L	LC <sub>50</sub> , 48-hour exposure
	Brown shrimp ( <i>Penaeus aztecus</i> )					0.40 µg/L	LC <sub>50</sub> , 48-hour exposure
	Natural phytoplankton communities			1,000 µg/L	28.5% decrease in productivity C-14		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
	Alga ( <i>Acetabularia mediterranea</i> )			10,000 µg/L	Inhibition of cell growth and cell morphogenesis; reversible		
	American oyster ( <i>Crassostrea virginica</i> )					450 µg/L* (mean for species—450 µg/L)	EC <sub>50</sub> : *decreased shell growth; flow-through, unmeasured
	Polychaete ( <i>Neanthes arenaeodentata</i> )					3,680 µg/L (mean for species—3,680 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, measured
	American eel ( <i>Anguilla rostrata</i> )					56 µg/L (mean for species—56 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, unmeasured
	Mummichog ( <i>Fundulus heteroclitus</i> )					60 µg/L (mean for species—60 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, unmeasured
	Striped killifish ( <i>Fundulus majalis</i> )					28 µg/L (mean for species—28 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, unmeasured
	Atlantic silverside ( <i>Menidia menidia</i> )					9 µg/L (mean for species—9 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, unmeasured
	Bluehead ( <i>Thalassoma bifasciatum</i> )					14 µg/L (mean for species—14 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, unmeasured
	Striped mullet ( <i>Mugil cephalus</i> )					66 µg/L (mean for species—66 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, unmeasured
Lindane, technical (EPA, 1980b)	Northern puffer ( <i>Sphaeroides maculatus</i> )					35 µg/L (mean for species—35 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static unmeasured
Benzene (IARC, 1982)	Rats, guinea-pigs, rabbits			264-291 mg/m <sup>3</sup> , inhal	Exposure of 7 hours/day for 30-40 weeks resulted in an increase in testicular weight and degeneration of seminiferous tubules.		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
	Rats			5.3-4.6 mg/m <sup>3</sup> , inhal	Alteration of estrous cycles, but no effect on subsequent fertility or litter size		
Benzene (ACGIH, 1986)	Rats	169-102 mg/m <sup>3</sup> , inhal	5-8 weeks exposure of 5 hours/day, 5 days/week, no effects	145 & 155 mg/m <sup>3</sup> , inhal	5-8 weeks exposure of 5 hours/day, 5 days/week, moderate degree of leukopenia.		
				66 mg/m <sup>3</sup> , inhal	756 hours exposure of 8 hours/day, 5 days/week, decrease in white cell count.		
Benzene (Keller, 1986)	Swiss-Webster mice			66 mg/m <sup>3</sup> , inhal	Exposed from days 6-15 of gestation. effects of fetuses include: decreased number of mature erythroid precursor cells, increased number of CFU-E cells, affected granulocytic colony forming cells.		
Benzene (Ballou et al., 1985)	Blue crab (juveniles)			0.4 mg/L	Increase in time needed to complete a molt cycle (50 days vs. 33 days for control), slower rate of limb bud regeneration, and depressed activity of ATPase in mitochondria.		
				3.3 mg/m <sup>3</sup> , inhal	Decrease in oxygen consumption		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
Benzene (Nawrot and Staples, 1979)	Rats	0.3-1.0 mg/kg body weight, diet	No teratogenic effects, but reduced fetal weight and occasional embryoletality.				
Benzene (Gofmekler, 1968)	Rats			209.7 g/kg, diet	Exposure 10 days before breeding showed a complete absence of pregnancy.		
Benzene (Buikema and Hendricks, 1980)	Algae			10 mg/L	Inhibition threshold for most marine algae	741 mg/L	<i>Anacharis canadensis</i> died after 1-hour exposure
Benzene (Verschuere, 1983)	Herring and anchovy larvae			10-35 mg/L	Delays in development of larvae, reduction in feed intake and growth		
	Grass shrimp ( <i>Palaemonetes pugio</i> )					27 mg/L/96 hours	LC <sub>50</sub>
	Crab larvae, Stage 1 ( <i>Cancer magister</i> )					108 mg/L/96 hours	LC <sub>50</sub>
	Shrimp ( <i>Crangon franciscorum</i> )					20 mg/L/96 hours	LC <sub>50</sub>
	Mexican axolotl (3-4 weeks after hatching) ( <i>Ambystoma mexicanum</i> )					370 mg/L/48 hours	LC <sub>50</sub>
	Clawed toad (3-4 weeks after hatching)					190 mg/L/48 hours	LC <sub>50</sub>
	Brine shrimp					21-66 mg/L/24 hours	LC <sub>50</sub>
Benzene (EPA, 1980c)	Pacific oyster ( <i>Crassostrea gigas</i> )					924,000 µg/L (mean for species—924,000 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static, un-measured
	Copepod ( <i>Tigriopus californicus</i> )					450,000 µg/L (mean for species—450,000 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static un-measured

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
	Bay shrimp ( <i>Crangon franciscorum</i> )					17,600 µg/L (mean for species—17,600 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static un-measured
	Grass shrimp ( <i>Palaemonetes pugio</i> )					27,000 µg/L (mean for species—27,000 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static un-measured
	Dungeness crab, larva ( <i>Cancer magister</i> )					108,000 µg/L (mean for species—108,000 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static un-measured
	Striped bass ( <i>Morone saxatilis</i> )			6,000 µg/L/168 hours	Temporary weight reduction	10,900 µg/L	LC <sub>50</sub> /EC <sub>50</sub> ; flow-through, measured
						5,100 µg/L (mean for species—10,900 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; flow-through, measured
	Copepod ( <i>Nitocra spinipes</i> )					82,000 µg/L	LC <sub>50</sub> ; 24-hour exposure
						111,500 µg/L	LC <sub>50</sub> ; 24-hour exposure
	Grass shrimp, adult ( <i>Palaemonetes pugio</i> )					38,000 µg/L	LC <sub>50</sub> ; 24-hour exposure
						33,500 µg/L	LC <sub>50</sub> ; 24-hour exposure
						40,200 µg/L	LC <sub>50</sub> ; 24-hour exposure
	Grass shrimp, larva ( <i>Palaemonetes pugio</i> )					40,800 µg/L	LC <sub>50</sub> ; 24-hour exposure
						90,800 µg/L	LC <sub>50</sub> ; 24-hour exposure
						74,400 µg/L	LC <sub>50</sub> ; 24-hour exposure
	Pacific herring ( <i>Clupea harengus pallasi</i> )			700 µg/L/144 hours	Stress observed		
						700 µg/L/168 hours	Survival reduction
	Dinoflagellate ( <i>Amphidinium carterae</i> )			>5,000 µg/L	Growth inhibition		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
	Diatom ( <i>Skeletonema costatum</i> )			100,000 µg/L	Growth inhibition		
	Diatom			20,000 µg/L	Growth inhibition		
Chlorobenzene (Kluwe, 1985)	F344/N rats					500 mg/kg, oral	91-day subchronic exposure reduced survival developed hepatocyte necrosis, necrosis of renal proximal tubules, lymphoid or myeloid depletion of the spleen, bone marrow, and thymus.
	B6C3F1 mice					250 mg/kg, oral	91-day subchronic exposure reduced survival developed hepatocyte necrosis, necrosis of renal proximal tubules, lymphoid or myeloid depletion of the spleen, bone marrow, and thymus.
Chlorobenzene (Clayton, 1981)	Rats	14.4 mg/kg/day, oral	5 days/week exposure for 192 days did not produce any observable effects.	0.144 g/kg body weight/day, oral	5 days/week exposure for 192 days produced transient growth retardation.		
				0.1444 & 0.288 g/kg body weight/day, diet	5 days/week exposure for 192 days resulted in significant increase in liver and kidney weight.		
Chlorobenzene (John et al., 1984)	Rabbits (6-18 days gestation)	2809 mg/m <sup>3</sup> , inhal	6 hours/day for 13 days, no developmental effects.				
	Rat (6-15 days gestation)	2809 mg/m <sup>3</sup> , inhal	6 hours/day for 10 days, no developmental effects				

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
Chlorobenzene (EPA, 1980d)	Mysid shrimp ( <i>Mysidopsis bahia</i> )					16,400 µg/L (mean for species—16,400 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static, unmeasured
	Sheepshead minnow ( <i>Cyprinodon variegatus</i> )					10,500 µg/L (mean for species—10,500 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static unmeasured
	Alga ( <i>Skeletonema costatum</i> )					343,000 µg/L	96 hour EC <sub>50</sub> ; Chlorophyll A
						341,000 µg/L	96 hour EC <sub>50</sub> ; reduction in cell number
Chloroform (Amdur et al., 1980)	Rats			1.05 ml/kg, diet	Single exposure resulted in 40% inhibition of microsomal drug metabolizing enzyme activity.		
	Mice	150-300 mg/kg body weight, oral	3 months old. Exposure to 30 oral doses produced no adverse affects.			0.6, 1.2, and 2.4 g/kg body weight, oral	Nonmetastasizing hepatomas and cirrhosis in male and female rats
Chloroform (Chu, 1980)	Rats					908 mg/kg, oral	LD <sub>50</sub> for male rats
						1117 mg/kg, oral	LD <sub>50</sub> for female rats
Chloroform (IARC, 1979)	Osborne-Mendel rats, males			90 and 180 mg/kg body weight, gavage	78 weeks exposure resulted in an increased incidence of kidney epithelial tumors.		
Chloroform (Moore, 1982)	Male, CFLP outbred Swiss Albino Mouse	18 mg/kg, oral	Single dose produced no detectable acute toxic effect on liver or kidneys and did not stimulate regenerative activity.	60 mg/kg, oral	Single dose resulted in toxicity and tissue regeneration in liver. Kidney tumors were also present.		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
Chloroform (Klaasen et al., 1986)	Rabbits, rats, guinea pigs, dogs			126, 253, or 429 mg/m <sup>3</sup> , inhal	Exposure of 7 hours/day, 5 days/week for 6 months resulted in centrilobular necrosis and cloudy swelling of kidneys. Symptoms from the 25 ppm exposure was mild and reversible.		
Chloroform (ACGIH, 1986)	Rats	126-152 mg/m <sup>3</sup> , inhal	Exposure of 7 hours/day, 5 days/week for 6 months did not produce organ injury	253 mg/m <sup>3</sup> , inhal	Exposure of 7 hours/day, 5 days/week for 6 months results in kidney injury. Severity of injury is concentration dependent.		
	Dogs	15 and 30 mg/kg/day, diet	No effects				
Chloroform (Larson, 1985)	White rat					2,180 mg/kg	LD <sub>50</sub> , oral
	Rabbit					9,827 mg/kg	LD <sub>50</sub> , oral
	Dog					2,250 mg/kg	LD <sub>50</sub> , oral
Chloroform (Chu, 1980)	Rat, male					908 mg/kg	LD <sub>50</sub> , oral
	Rat, female					1,117 mg/kg	LD <sub>50</sub> , oral
Chloroform (EPA, 1980e)	Pink shrimp					81,500 µg/L/96 hours	LC <sub>50</sub> , static bioassay
1,2-Dichloroethane (Shepard, 1986)	Rats	250-500 mg/kg, diet	2-year exposure, approx. 60-70% of dose consumed. No significant decrease in fertility, litter size, or fetal weight was observed.				

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
		418.6 mg/m <sup>3</sup> , inhal	Exposed 7 hours/day during days 6-15 of gestation. No resorption or decrease in fetal weight.			1,256 mg/m <sup>3</sup> , inhal	Exposed 7 hours/day during days 6-15 of gestation. Ten of 16 rats died.
1,2-Dichloroethane (Rao et al., 1980)	Rabbit (6-18 days gestation)	1,256 mg/m <sup>3</sup> , inhal	Exposed 7 hours/day (duration not specified). no developmental effects.				
1,2-Dichloroethane (Lane, 1982)	ICR Swiss mice	0, 0.03, 0.09, or 0.29 mg/ml in water, diet	No dose-dependent effects on fertility, gestation, viability, or lactation indices. The survival of the pups and weight gain were not adversely affected.				
1,2-Dichloroethane (Larson, 1985)	Mouse					870-950 mg/kg	LD <sub>50</sub> , oral
	Rabbit					860-970 mg/kg	LD <sub>50</sub> , oral
1,2-Dichloroethane (Worthing, 1987)	Rat					670-890 mg/kg	LD <sub>50</sub> , oral
1,2-Dichloroethane (Verschuere, 1983)	Rat					50,232 mg/m <sup>3</sup> /31.8min 12,558 mg/m <sup>3</sup> /165min 4,186 mg/m <sup>3</sup> /432min	LC <sub>50</sub>
	Sand shrimp ( <i>Crangon crangon</i> )					75 mg/L/24 hours, and 65 mg/L/96 hours	LC <sub>50</sub> , in seawater
	Gobi					185 mg/L/60 minutes	LC <sub>50</sub> , in seawater
1,2-Dichloroethane (Kayser, 1982)	Mysid shrimp ( <i>Mysidopsis bahia</i> )					113,000 µg/L/96 hours	LC <sub>50</sub> , in saltwater
1,2-Dichloroethane (EPA, 1980f)	Mysid shrimp ( <i>Mysidopsis bahia</i> )					113,000 µg/L (mean for species—113,000 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static, unmeasured bioassay

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
	Alga ( <i>Skeletonema costatum</i> )					>433,000 µg/L	95 hour EC <sub>50</sub> , Chlorophyll A
						>433,000 µg/L	96 hour EC <sub>50</sub> , reduction in cell count
Ethylbenzene (Verschuere, 1983)	Coho salmon					50 mg/L	100% mortality in young salmon after 24-hour exposure
						10 mg/L	2 out of 30 died after 24 and 96 hours exposure
	Rat					3,500 mg/kg, oral	LD <sub>50</sub> , single dose
Ethylbenzene (Cragg et al., 1989)	Rat	3,512 mg/m <sup>3</sup> , inhal	5 days/week, 6 hours/day, 4 weeks; no reproductive effects				
	Mouse	3,512 mg/m <sup>3</sup> , inhal	5 days/week, 6 hours/day, 4 weeks; no reproductive effects				
Ethylbenzene (Andrew et al., 1981)	Rabbit (1-24 days gestation)	4,491 mg/m <sup>3</sup> , inhal	Exposure for 24 days, 7 hours/day, no developmental effects				
Ethylbenzene (Clayton, 1981)	Rats			408-680 mg/kg/day, oral	182-day exposure results in liver and kidney weight increase.		
Ethylbenzene (Clayton, 1981)	Rabbits, guinea pigs, and monkey	1,796-8,981 mg/m <sup>3</sup> , inhal	No observed affects.				
Ethylbenzene (EPA, 1978)	Shrimp					87.6 mg/L/96 hours	LC <sub>50</sub> , static unmeasured bioassay
	Grass shrimp, adult					14,440 µg/L/24 hours	LC <sub>50</sub> , static unmeasured bioassay
	Grass shrimp, larva					10,200 µg/L/24 hours	LC <sub>50</sub> , static unmeasured bioassay

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
Ethylbenzene (EPA, 1980g)	Pacific oyster ( <i>Crassostrea gigas</i> )					1,030,000 µg/L (mean for species—1,030,000 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, un-measured
	Bay shrimp ( <i>Crangon franciscorum</i> )					3,700 µg/L (mean for species—3,700 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, measured
	Mysid shrimp ( <i>Mysidopsis bahia</i> )					87,600 µg/L (mean for species—87,600 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, un-measured
	Sheepshead minnow ( <i>Cyprinodon variegatus</i> )					275,000 µg/L (mean for species—275,000 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, un-measured
	Striped bass ( <i>Morone saxatilis</i> )					430 µg/L (mean for species—430 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static un-measured
	Alga ( <i>Skeletonema costatum</i> )					>438,000 µg/L	96 hours EC <sub>50</sub> for Chlorophyll A
Ethylbenzene (Ballou et al., 1985)	Manila clams ( <i>Tapes semidecussata</i> )			0.08 mg/L	Increased mucous production		
Toluene (ACGIH, 1986)	Mice			11,693 mg/m <sup>3</sup> , inhal	Prostration	38,975 mg/m <sup>3</sup> , inhal	Acute poisoning
	Rats			9,744-19,488 mg/m <sup>3</sup> , inhal	Temporary decrease in white cell count—no injuries to blood forming organs.		
Toluene (Clayton, 1981)	Cats			30,401 mg/m <sup>3</sup> , inhal	6 hours exposure produced CNS effects, mydriasis, mild tremors, prostration in 80 minutes. Light anesthesia in 2 hours.		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
Toluene (Shepard, 1986)	Rats	1,500 mg/m <sup>3</sup> , inhal	Daily exposure on day 1-8 produced no teratogenic effect, some fetal growth retardation				
		1,000 mg/m <sup>3</sup> , inhal	8 hours daily exposure from day 1-21 produced no teratogenic effect, some fetal growth retardation				
	Mice	1,500 mg/m <sup>3</sup> , inhal	8 hours daily exposure in days 6-15 produced no teratogenic effect, some fetal growth retardation				
				1.0 mg/kg, gavage	Exposure on days 6-15 of gestation increased cleft palate in offspring.		
Toluene (Ungvary and Tatrai, 1985)	Rabbit	518 mg/m <sup>3</sup> , inhal	24 hours/day, 14 days; no developmental effects	267 mg/kg	Fetus aborted		
Toluene (API, 1981)	Mouse	1,559 mg/m <sup>3</sup> , inhal	5 days/week, 6 hours/day, 8 weeks; no reproductive effects				
Toluene (Buikema and Hendricks, 1980)	Chlorella					132.5 mg/m <sup>3</sup>	Growth inhibition (based on photosynthesis/ respiration ratio)
Toluene (WHO, 1985)	Marine algae ( <i>Chlorella vulgaris</i> )			245 mg/L	EC <sub>50</sub> , 24-hour exposure		
	Marine algae ( <i>Selenastrum capri cornutum</i> )			>433 mg/L	EC <sub>50</sub> , 96-hour exposure		
	Zebra fish					25 mg/L	LC <sub>50</sub> , 48-hour exposure
	Salmon fry (coho)					5.5 mg/L	LC <sub>50</sub> , 96-hour exposure
	Salmon fry (pink)					7 mg/L	LC <sub>50</sub> , 96-hour exposure

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
	General aquatic organisms			2 mg/L	Ambient water concentration. Inhibited reproduction and decreased growth rates		
				8 mg/L	Narcosis in sea water		
	Grass shrimp ( <i>Palaemonetes pugio</i> )					17.2 mg/L	LC <sub>50</sub> , 24-hour exposure
	Brine shrimp					33 mg/L	LC <sub>50</sub> , 24-hour exposure
	Bay shrimp					3.7 mg/L	LC <sub>50</sub> , 96-hour exposure
	Dungeness crab					170 mg/L	LC <sub>50</sub> , 48-hour exposure
Toluene (Verschuereen, 1983)	Grass shrimp ( <i>Palaemonetes pugio</i> )					48 mg/L	LC <sub>50</sub> , 96-hour exposure
						9.6 mg/L/96 hours	LC <sub>50</sub>
	Dungeness crab larvae, Stage 1					28 mg/L/96 hours	LC <sub>50</sub>
	Bay Shrimp ( <i>Crangon franciscorum</i> )					4.3 mg/L/96 hours	LC <sub>50</sub>
Toluene (Prince, 1974)	Brine Shrimp					33 mg/L/24 hours	LC <sub>50</sub>
Toluene (Ferguson and Pirie, 1948)	Grain Weevil					210 mg/L	LD <sub>50</sub> , in air
Toluene (ASTER, 1992)	Coho salmon ( <i>Oncorhynchus kisutch</i> )	1,410 µg/L	Reduction in growth at 40 days				
		1,650 µg/L	Avoidance at 1 hour				
Toluene (EPA, 1980h)	Pacific oyster ( <i>Crassostrea gigas</i> )					1,050,000 µg/L (mean for species—1,050,000 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static, un-measured
	Mysid shrimp ( <i>Mysidopsis bahia</i> )					56,300 µg/L (mean for species—56,300 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> ; static, un-measured

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
	Bay shrimp ( <i>Crangon franciscorum</i> )					3,700 µg/L (mean for species—3,700 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, un-measured
	Grass shrimp ( <i>Palaemonetes pugio</i> )					9,500 µg/L (mean for species—9,500 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, un-measured
	Striped bass ( <i>Morone saxatilis</i> )					6,300 µg/L (mean for species—6,300 µg/L)	LC <sub>50</sub> /EC <sub>50</sub> : static, un-measured
	Copepod ( <i>Nitocra spinipes</i> )					24,200 µg/L	LC <sub>50</sub> , 24-hour exposure
						74,200 µg/L	LC <sub>50</sub> , 24-hour exposure
	Grass shrimp, adult ( <i>Palaemonetes pugio</i> )					20,200 µg/L	LC <sub>50</sub> , 24-hour exposure
						17,200 µg/L	LC <sub>50</sub> , 24-hour exposure
						37,600 µg/L	LC <sub>50</sub> , 24-hour exposure
						38,100 µg/L	LC <sub>50</sub> , 24-hour exposure
	Grass shrimp, larva ( <i>Palaemonetes pugio</i> )					30,600 µg/L	LC <sub>50</sub> , 24-hour exposure
						25,800 µg/L	LC <sub>50</sub> , 24-hour exposure
	Grass shrimp ( <i>Palaemonetes pugio</i> )					19,800 µg/L	Narcosis
	Coho salmon ( <i>Oncorhynchus kisutch</i> )					10,000—50,000 µg/L	LC <sub>50</sub> , 96-hour exposure
	Sheepshead minnow ( <i>Cyprinodon variegatus</i> )					>277,000— <485,000 µg/L	LC <sub>50</sub> , 96-hour exposure
	Kelp ( <i>Macrocystis pyrifera</i> )			10,000 µg/L	Photosynthesis inhibition		
	Alga ( <i>Amphidinium carteri</i> )			100,000 µg/L	Growth inhibition		
	Alga ( <i>Chlorella sp.</i> )			34,000 µg/L	Photosynthesis and respiration inhibition		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
				100,000 µg/L	Growth inhibition		
	Alga ( <i>Dunaliella tertiolecta</i> )			100,000 µg/L	Growth inhibition		
	Alga ( <i>Skeletonema costatum</i> )			8,000 µg/L	Growth inhibition		
						>433,000 µg/L	96 hours EC <sub>50</sub> for Chlorophyll A production
						>433,000 µg/L	96 hours EC <sub>50</sub> for reduction in cell numbers
Toluene (Ballou et al., 1985)	Pacific oyster ( <i>Crassostrea gigas</i> )			3,100 µg/L	Reduced developmental growth		
	California mussel ( <i>Mytilus californianus</i> )			100,000 µg/L	Reduced respiration and heart rate		
	Manila clam ( <i>Tapes semidecussata</i> )			1,300 µg/L	Increased mucous production		
Xylenes (Shepard, 1986)	Rats	1,000 mg/m <sup>3</sup> , inhal	Exposure during days 9-14 of pregnancy produced no teratogenic results, although minor skeletal anomalies occurred.				
Xylenes (Muralidhara and Krishnakumari 1980)	Female Wistar CFT rats	6 ml/kg, diet	Not lethal			7 ml/kg	Minimum lethal dose.
Xylenes (Mirkova, 1983)	Rats			50 mg/m <sup>3</sup> , inhal	Embryotoxic and teratogenic effects. brain, liver, lung, heart effected. Post-implantation losses increased by 9.7%. Incidence of fetal skeletal abnormalities increased 62%.		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
				500 mg/m <sup>3</sup> , inhal	Embryotoxic and teratogenic effects. Brain, liver, lung, heart effected. Post-implantation losses increased by 168%. Incidence of fetal skeletal abnormalities increased 177%.		
Xylenes (David, 1982)	Quail eggs					2 or 0.05% aqueous suspensions	Direct spraying of shell or repeated ingestion by parent quail reduced hatching rate, embryonic viability, increased fecundation rate, and weight of eggs, chickens, and adults.
Xylenes (NTP, 1986)	F344/N rats and B6C3F1 mice					6,000 mg/kg, gavage	Death in male and female rats and mice.
						4,000 mg/kg, gavage	Death in male rats
	Rats	1,000 mg/kg, gavage	No deaths or clinical signs of toxicity.	2,000 mg/kg, gavage	Males gained 15% less weight, and females gained 8% less weight than controls. Symptoms include lethargy, short, and shallow breathing, unsteadiness, tremors, and paresis. Effects lasted 15-60 min.		
Xylenes (Hudak and Ungvary, 1978)	Rat (9-14 days gestation)			1,033 mg/m <sup>3</sup> , inhal	LOAEL; 24 hours/day, 6 days exposure resulted in increased fetal anomalies		

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
Xylenes (Ungvary and Tatrai, 1985)	Rat (7-15 days gestation)			260 mg/m <sup>3</sup> , inhal	LOAEL; 24 hours/day, 9 days exposure resulted in skeletal retardations		
				3,521 mg/m <sup>3</sup> , inhal	increased fetal death and resorption		
Xylenes (Verschuere, 1983)	Algae ( <i>Chlorella Vulgaris</i> )					55 mg/L	Exposure to ortho-xylene decreased cell number by 50%.
	Grass shrimp ( <i>Palaemonetes pugio</i> )					7.4 mg/L	96 hours LC <sub>50</sub> (o-xylene)
	Crab larvae—Stage 1 ( <i>Cancer magister</i> )					6.0 mg/L 12 mg/L	(o-xylene) 96 hours LC <sub>50</sub> (m-xylene) (m-xylene)
	Bay shrimp ( <i>Crangon franciscorum</i> )					1.3 mg/L 3.7 mg/L 2.0 mg/L	(o-xylene) (m-xylene) (p-xylene) all 96-hour LC <sub>50</sub> s
	Young Coho salmon ( <i>Oncorhynchus kisutch</i> )					10.0 mg/L	(o-xylene) no mortality in 96 hours
	Striped bass ( <i>Morone saxatilis</i> )					11.0 mg/L 9.2 mg/L 2.0 mg/L	(o-xylene) 96 hours or less (m-xylene) 96-hour LC <sub>50</sub> (p-xylene) 96-hour LC <sub>50</sub>
Xylenes (Ballou et al., 1985)	Pacific oyster ( <i>Crossostrea gigas</i> ) (larvae)			3.1 mg/L	Altered development		
				1.22 mg/L	Altered development m-xylene		
				0.359 mg/L	Altered development o-xylene		
				0.359 mg/L	Altered development p-xylene		

Continued

**Appendix B**  
**Toxicity Profile for Contaminants of Concern**

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Element (reference)	Species	Effects Documented					
		No Observed Effect		Nonlethal Effect		Reduced Survival	
		Concentration	Comment	Concentration	Comment	Concentration	Comment
	Manila clam ( <i>Tapes semidecussata</i> )			0.130 mg/L	Increased mucous production (o-xylene)		
Xylenes (Hudak and Ungvary, 1978)	CFY rats			1,000 mg/m <sup>3</sup> , inhalation	Exposure of pregnant rats on gestation days 9-14 produced skeletal effects including an increase incidence of supernumerary ribs.		
Xylenes (Clayton, 1981)	Rat					4.3 g/kg	LD <sub>50</sub> , oral
						28,518 mg/m <sup>3</sup> /4 hours	LC <sub>50</sub>
Xylenes (NRC, 1981)	Mouse					17,546 mg/m <sup>3</sup> /6 hours	LC <sub>50</sub>
<sup>a</sup> Technical BHC (21% alpha BHC, 2.1% beta BHC, 39% gamma BHC, 23% delta BHC, 14.9% unidentified compounds)							

## References for Appendix B

### Toxicity Profile for Contaminants of Concern

ACGIH. 1986. Documentation of TLVS.: 5th ed. As cited in TOXNET, Hazardous Substances Data Bank.

ATSDR. 1989a. Toxicology Profile for Chloroform. National Technical Information Service PB89-160360. January.

\_\_\_\_\_. 1989b. Toxicology Profile for Benzene. National Technical Information Service PB89-209464. May.

\_\_\_\_\_. 1989c. Toxicological Profile for DDT, DDE, and DDD. U.S. Public Health Service Contract No. 205-88-0608.

\_\_\_\_\_. 1989d. Toxicology Profile for 1, 2-Dichloroethane. National Technical Information Service PB90-171422. December.

\_\_\_\_\_. 1989e. Toxicology Profile for Toluene. National Technical Information Service PB90-198904. January.

\_\_\_\_\_. 1989f. Alpha-, Beta-, Gamma-, and Delta-hexachlorocyclohexane. National Technical Information Service PB90-171406. December.

\_\_\_\_\_. 1990a. Toxicological Profile for Chlorobenzene. U.S. Department of Health and Human Services, Washington, D.C., National Technical Information Service PB91-180505. December.

\_\_\_\_\_. 1990b. Toxicology Profile for Ethylbenzene. U.S. Department of Health and Human Services, Washington, D.C., National Technical Information Service PB91-180372. December.

\_\_\_\_\_. 1990c. Toxicology Profile for Total Xylenes. U.S. Department of Health and Human Services, Washington, D.C., National Technical Information Service PB91-181552. December.

Amdur, M.O.; Doull, J.; Klaassen, C.D., editors. 1980. Casarett and Doull's Toxicology, Second edition. Pergamon Press, Inc. As cited in TOXNET, Hazardous Substances Data Bank.

Andrew et al. 1981. As cited in ATSDR, 1990b.

API. 1981. As cited in ATSDR, 1989c.

ASTER Ecotoxicity Profile. 1992. EPA Environmental Research Laboratory. Duluth.

Ballou, T.G., et al. 1985. Summary and Evaluation of THR Toxicological and physiological effects of pollutants on shellfish. Part Two (Petroleum Hydrocarbons) and Part Three (Pesticides). NOAA, Office of Marine Pollution Assessment, Office of Operational Programs RD/MP2. Seattle, Washington.

Buikema, A.L., Jr. and A.C. Hendricks. 1980. Benzene, Xylene, and Toluene in Aquatic Systems: A Review. Washington, D.C.: American Petroleum Institute.

Cabral, J.R.P. et al. 1982. Tumori. 68 (1):11-8. As cited in TOXNET, Hazardous Substances Data Bank.

Cantelmo, A. et al. 1982. Physiol. Mech. Mar. Pollut. Toxic. 349-89. As cited in TOXNET, Hazardous Substances Data Bank.

Chu, I. et al. 1980. Toxicol. Appli. Pharmacology. 52 (2):351-3. As cited in TOXNET, Hazardous Substances Data Bank.

Clarke. 1981. Vet. Tox. 140 pp. As cited in TOXNET, Hazardous Substances Data Bank.

Clayton, G.D., and F.E. Clayton. 1981. Patty's Industrial Hygiene and Toxicology. 3rd ed. Vol. 2A, 2B, 2C. John Wiley & Sons. As cited in TOXNET, Hazardous Substances Data Bank.

Cragg et al. 1989. As cited in ATSDR, 1990a.

David, D. 1982. Arch. Anat. Microsc. Morphol. Exp. 71(3):159-74. As cited in TOXNET, Hazardous Substances Data Bank.

Davison, K.L., and J.L. Sell. 1974. DDT thins shells of eggs from mallard ducks maintained on ad libitum or controlled-feeding regimens. Arch. Environ. Contam. Toxicol. 2:222-232.

DeWaziers, I. and V. Azias. 1987. Drug-metabolizing enzyme activities in the liver and intestine of rats exposed to DDT: effects of Vitamin A status. Arch. Environ. Contam. Toxicol. 16:343-348.

DHEW. 1978. Bioassays of DDT, TDE, & p,p-DDE for Possible Carcinogenicity. Report #TR-131. DHEW Pub No. 78-1386. As cited in TOXNET, Hazardous Substances Data Bank.

Fabro, S. et al. 1984. Am. J. Obstet. Gynecol. 148 (87):929-38. As cited in TOXNET, Hazardous Substances Data Bank.

Ferguson, J., and H. Pirie. 1948. *Ann. Appl. Biol.* 35:532-550. As cited in National Research Council (NRC). 1981. Committee on Alkyl Benzene Derivatives. *The Alkyl Benzenes*. Washington, D.C.: National Academy Press. 363 pp. As cited in TOXNET, Hazardous Substances Data Bank.

Ferguson, D.E., and C.C. Gilbert. 1967. *J. Miss. Acad. Sci.* 13:135. As cited in USEPA Ambient Water Quality Criteria Doc: DDT. 1980. Report No. EPA 440/5-80-038. B-48 pp. As cited in TOXNET, Hazardous Substances Data Bank.

Ferrando, M.D. and Andreu-Moliner. 1991. Acute lethal toxicity of some pesticides to *Brachionus calyciflorus* and *Brachionus plicatilis*. *Bull. Environ. Contam. Toxicol.* 47:479-484.

Ferrando, M.D. et al. 1989. Short-term lindane effects on gill tissue metabolism of the eel. *Toxicol. Environ. Chem.* 24:17-23.

Fitzhugh et al. 1950. IARC Monographs 1972—present. 1979. 20:214. As cited in TOXNET, Hazardous Substances Data Bank.

Gish, C.D. and N.J. Chura. 1970. Toxicity of DDT to Japanese quail as influenced by body weight, breeding condition, and sex. *Toxicol. Appl. Pharmacol.* 17:740-751.

Gofmekler, V.A. 1968. *Hyg. Sanit.* 33:327. As cited in TOXNET, Hazardous Substances Data Bank.

Goto et al. 1972. IARC Monographs 1972—present. 1979. 20:208. As cited in TOXNET, Hazardous Substances Data Bank.

Hanada et al. 1973. IARC Monographs 1972—present. 1979. 20:211. As cited in TOXNET, Hazardous Substances Data Bank.

Hart et al. 1972. As cited in ATSDR, 1989b.

Hartley. 1987. *Agrochemical Handbook*. Second Edition. 118 pp. As cited in TOXNET, Hazardous Substances Data Bank.

Heath, R.G., J.W. Spann, and J.F. Kreitzer. 1969. Marked DDE impairment of mallard reproduction in controlled studies. *Nature* 224 (5214):47-48.

Heinz, G.H. 1976. Behavior of mallard ducklings from parents fed 3 ppm DDE. *Bull. Environ. Contam. Toxicol.* 16:640-5. As cited in TOXNET, Hazardous Substances Data Bank.

Hill, E.F., and M.B. Camardese. 1986. Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to Coturnix. U.S. Fish and Wildlife Service. Fish and Wildlife Technical Report 2. Washington, D.C.

Hill, E.F. et al. 1975. Lethal Dietary Toxicities of Environmental Pollutants to Birds. U.S. Fish and Wildlife Service. Spec. Sci. Rep. Wildl. 191. 61 pp.

Hudak, A., and G. Ungvary. 1978. Toxicol. 11:55-63. As cited in TOXNET, Hazardous Substances Data Bank.

Hudson, R.H., R.K. Tucker, and M.A. Haegele. 1984. Handbook of Toxicity of Pesticides to Wildlife. Second edition.. U.S. Dept. of Interior. Fish and Wildlife Service. Resour. Pub. 153. Washington, D.C.

IARC. 1974. Monographs 1972—present. 5:106. As cited in TOXNET, Hazardous Substances Data Bank.

IARC. 1979. Monographs 1972—present, 20:411. As cited in TOXNET, Hazardous Substances Data Bank.

IARC. 1982. Monographs 1972—present. 29:111. As cited in TOXNET, Hazardous Substances Data Bank.

Ito et al. 1973. IARC Monographs 1972—present. 1979. 20:212. As cited in TOXNET, Hazardous Substances Data Bank.

Jefferies, D.J. 1971. Some sublethal effects of p,p'-DDT and its metabolites p,p'-DDE on breeding passerine birds. Med. Fak. Landb. Wentenschappen Gent. 36:34-42.

Jensen, L.D., and A.R. Gaufin. 1964. Trans. Amer. Fish. Soc. 93:357. As cited in USEPA Ambient Water Quality Doc: DDT. B-45 pp. As cited in TOXNET, Hazardous Substances Data Bank.

John et al. 1984. As cited in ATSDR, 1990a.

Johnson, W.W., and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. U.S. Fish and Wildlife Service. Resour. Pub. 137. Washington, D.C.

Kayser. 1982. Inter. Priority Pollut. As cited in TOXNET, Hazardous Substances Data Bank. 1980. Report No. EPA 440/5-80-038.

Keller, K.A., and C.A. Snyder. 1986. Toxicology 42:171-81. As cited in TOXNET, Hazardous Substances Data Bank.

Klaasen, C.D., M.O. Amdur, and J. Doull. 1986. Casarett and Doull's Toxicology, Third edition. Macmillan Publishing Company. As cited in TOXNET, Hazardous Substances Data Bank.

Kluwe, W.M. et al. 1985. J. Toxicol. Environ. Health. 15 (6):745-67. As cited in TOXNET, Hazardous Substances Data Bank.

Kornburst, D. et al. 1986. J. Toxicol. 17 (1):23-36. As cited in TOXNET, Hazardous Substances Data Bank.

Lane, R.W. et al. 1982. Toxicol. Appl. Pharmacol. 63:409-21. As cited in TOXNET, Hazardous Substances Data Bank.

Larson. 1985. Commercial & Experimental Organic Insecticides. 85 REV. 26 pp. As cited in TOXNET, Hazardous Substances Data Bank.

Longcore, J.R., and F.B. Samson. 1973. Eggshell breakage by incubating black ducks fed DDE. J. Wildl. Manage. 37:390-394.

Longcore, J.R. et al. 1971. Changes in mineral composition of eggshells from black ducks and mallards fed DDE in the diet. Bull. Environ. Contam. Toxicol. 6:345-350.

Lowe, J.I. et al. 1971. Chronic exposure of oysters to DDT, toxaphene and parathion. Proc. Nat. Shellfisheries Assoc. 61:71-79.

Ludke, J.L. 1974. Interaction of dieldrin and DDE residues in Japanese quail (*Coturnix coturnix japonica*). Bull. Environ. Contam. Toxicol. 11:297-302.

Macek, K.J., and W.A. McAllister. 1970. Trans. American Fish Soc. 99:89. As cited in USEPA Ambient Water Quality Criteria Doc: DDT. 1980. Report No. EPA 440/5-80-038. B-45 pp. As cited in TOXNET, Hazardous Substances Data Bank.

Matsumura. 1985. Toxicity of Insecticides. Second edition. As cited in TOXNET, Hazardous Substances Data Bank.

McLeese, D.W., and C.D. Metcalfe. 1980. Toxicities of eight organochlorine compounds in sediments and seawater to *Crangon septemspinosa*. Bull. Environ. Contam. Toxicol. 45 (6):921-928. As cited in ASTER Ecotoxicity Profile, 1992, EPA Environmental Research Laboratory, Duluth.

Meister, Richard T. editor. 1989. Farm Chemicals Handbook. Meister Publishing Co. As cited in TOXNET, Hazardous Substances Data Bank.

Meith-Avcin, N. 1974. DDT and the rugophilic response of settling barnacles *Balanus improvisus*. J. Fish. Res. Board Can. 31:1960-1963.

Miller, D.S. et al. 1976. DDE feeding and plasma osmoregulation in ducks, guillemots and puffins. Amer. J. of Physiol., 231 (2):370.

Mirkova, E. et al. 1983. J. Hyg. Epidemiol. Microbiol. Immunol. 27 (3):337-43. As cited in TOXNET, Hazardous Substances Data Bank.

Moore, D.H. et al. 1982. Food Chemistry Toxicology. 20 (6):951-4. As cited in TOXNET, Hazardous Substances Data Bank.

Muralidhara, and M.K. Krishnakumari. 1980. IND Journal Experimental Biology. 18:1148. As cited in TOXNET, Hazardous Substances Data Bank.

Nawrot and Staples. 1979. IARC Monographs 1972—present. 1982. 29:111. As cited in TOXNET, Hazardous Substances Data Bank.

NRC. 1981. The Alkyl Benzenes. As cited in Environment Canadian Technical Information for Problem Spills: Xylene (Draft). 1981. 81 pp. As cited in TOXNET, Hazardous Substances Data Bank.

NTP. 1986. Toxicology and Carcinogenesis Studies of Xylenes (Mixed). Report No. TR 327. NIH Publication No. 87-2583. As cited in TOXNET, Hazardous Substances Data Bank.

Odum et al. 1969.

Orberg and Lundberg. 1974. As cited in ATSDR, 1989c.

Pasha, S. 1981. As cited in ATSDR, 1989c.

Porter, R.D. and S.N. Wiemeyer. 1972. DDE at low dietary levels kills captive American kestrels. Bull. Environ. Contam. Toxicol. 8:193-199.

Powers, C.D., C.F. Wurster, and R.G. Rowland. 1975. The toxicity of DDE to a marine dinoflagellate. Environ. Pollut. 9:253-262.

Powers, C.D., C.F. Wurster, and R.G. Rowland. 1979. Pesticide Biochemistry and Physiology. 10:306-312.

Prince, K.S. et al. 1974. J. Water Pollut. Control Fed. 46:63-77. As cited in NRC; Alkyl Benzenes. p. 355. 1981. As cited in TOXNET, Hazardous Substances Data Bank.

Rao et al. 1980. As cited in ATSDR, 1989d.

Reddy, M.S., and K.V.R. Rao. 1986. Acute toxicity of insecticides to panacoid prawns. Environ. Ecol. 14:221-223.

Risebrough, R.W., and D.W. Anderson. 1975. Some effects of DDE and PCB on mallards and their eggs. J. Wildl. Manage. 39:508-513.

Rudolf, S. G. et al. 1984. Prey-capturing ability of American kestrel fed DDE and acephate or acephate alone. Arch. Environ. Contam. Toxicol. 13:367-372.

Sanders, H.O. 1970. Copeia. 2:246. As cited in USEPA; Ambient Water Quality Criteria Doc: DDT. 1980. p.B-49. EPA 440/5-80-038. As cited in TOXNET, Hazardous Substances Data Bank.

Schimmel, S.C., J.M. Patrick, and J. Forester. 1977. Toxicity and bioconcentration of BHC and lindane in selected estuarine animals. Arch. Environ. Contam. Toxicol. 6:355-363.

Shepard. 1986. CAT Teratogenic Agents. 5th ed. 169 pp. As cited in TOXNET, Hazardous Substances Data Bank.

Shivanandappa and Krishnakumari. 1983. As cited in ATSDR, 1989f.

Smith, S.I. et al. 1969. The effect of high levels of dietary DDT on egg production, mortality, fertility, hatchability, and pesticide content of yolks in Japanese quail. Poultry Sci. 48:1000-1004.

Stickel, W.H. et al. 1984. DDE in birds: lethal residues and loss rates. Arch. Environ. Contam. Toxicol. 13:1-6. As cited in TOXNET, Hazardous Substances Data Bank.

Tomatis, L. et al. 1974. Journal National Cancer Institute. 52 (3):883-91. As cited in TOXNET, Hazardous Substances Data Bank.

Ungvary and Tatrai. 1985. As cited in ATSDR, 1990c, 1989e.

EPA. 1978. In Depth Studies on Health and Environ. Impact of Sel. Water Poll.. EPA 68-01-4646. as cited in National Research Council (NRC). 1981. Committee on Alkyl Benzene Derivatives. The Alkyl Benzenes. Washington, D.C.: National Academy Press. 356 pp. As cited in TOXNET, Hazardous Substances Data Bank.

\_\_\_\_\_. 1980a. Ambient Water Quality Criteria Doc: DDT. Report No. EPA-440/5-80-038. Washington, D.C.: Criteria and Standards Division. Office of Water Regulations and Standards. As cited in ASTER Ecotoxicity Profile, EPA, Environmental Research Laboratory, Duluth.

\_\_\_\_\_. 1980b. Ambient Water Quality Criteria for Hexachlorocyclohexane. Report No. EPA-440/5-80-054. Washington, D.C.: Criteria and Standards Division. Office of Water Regulations and Standards. As cited in ASTER Ecotoxicity Profile, EPA, Environmental Research Laboratory, Duluth.

. 1980c. Ambient Water Quality Criteria for Benzene. Report No. EPA-440/5-80-018. Washington, D.C.: Criteria and Standards Division. Office of Water Regulations and Standards. As cited in ASTER Ecotoxicity Profile, USEPA, Environmental Research Laboratory, Duluth.

. 1980d. Ambient Water Quality Criteria for Chlorobenzene. Report No. EPA-440/5-80-028. Washington, D.C.: Criteria and Standards Division. Office of Water Regulations and Standards. As cited in ASTER Ecotoxicity Profile, EPA, Environmental Research Laboratory, Duluth.

. 1980e. Ambient Water Quality Criteria for Chloroform. Report No. EPA-440/5-80-033. As cited in TOXNET, Hazardous Substances Data Bank.

. 1980f. Ambient Water Quality Criteria for Chlorinated Ethanes. Report No. EPA-440/5-80-029. Washington, D.C.: Criteria and Standards Division. Office of Water Regulations and Standards. As cited in ASTER Ecotoxicity Profile, USEPA, Environmental Research Laboratory, Duluth.

. 1980g. Ambient Water Quality Criteria for Ethylbenzene. Report No. EPA-440/5-80-048. Washington, D.C.: Criteria and Standards Division. Office of Water Regulations and Standards. As cited in ASTER Ecotoxicity Profile, EPA, Environmental Research Laboratory, Duluth.

. 1980h. Ambient Water Quality Criteria for Toluene. Report No. EPA-440/5-80-075. Washington, D.C.: Criteria and Standards Division. Office of Water Regulations and Standards. As cited in ASTER Ecotoxicity Profile, EPA, Environmental Research Laboratory, Duluth.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals. Second edition. Van Nostrand Reinhold Co. NY. As cited in TOXNET, Hazardous Substances Data Bank.

Whitehead, C.C., J.N. Downie, and J.A. Phillips. 1974. Some characteristics of the egg shells of quail fed gamma-BHC. Pestic. Sci. 5:275-279.

WHO. 1985. As cited in TOXNET, Hazardous Substances Data Bank.

Worthing. 1987. Pesticide Manual. 8th ed. 358 pp. As cited in TOXNET, Hazardous Substances Data Bank.

Wurster, C.F. 1968. Science 159. 1474. As cited in TOXNET, Hazardous Substances Data Bank.